SQUINT

WORTH

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ITS CAUSES, PATHOLOGY, AND TREATMENT

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BY

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PREFACE TO THE FIRST EDITION.

By examining a very large number of cases of squint, and watching the results of treatment during a number of years, and by investigating the visual functions of normal-sighted people, I have endeavoured to learn the causes and pathology of squint. The methods of treatment which I employ are the outcome of these observations.

In cases of constant unilateral convergent squint, the usual routine treatment, by glasses and operation, gives extremely unsatisfactory results. In about one-third of these cases, the wearing of glasses causes the eyes, after a time, to become "straight." In the other two-thirds, the *deformity* may be more or less removed by operation. But, more often than not, the deviating eye becomes very blind, and the acquisition of any sort of binocular vision is quite the exception.

On the other hand, cases of unilateral squint in which treatment is commenced early and carried out by the methods described in these

pages are nearly always perfectly cured, having good vision in each eye, and good binocular vision.

Since 1893 I have kept detailed notes of every case of squint which has come under my observation. I have notes of 2,337 squints and heterophorias. Of these cases, 1,729 suffered from convergent squint. The cases presented themselves in the out-patient departments of the West Ham and East London Hospital, the Loughborough Hospital, and the Royal London Ophthalmic Hospital (Moorfields), and in my private practice.

I am greatly indebted to Mr. Silcock and Mr. Holmes Spicer for their kindness in allowing me, during a period of nearly four years, to investigate and treat the cases of squint attending their out-patient clinics at Moorfields.

I wish to thank Mr. R. E. Hanson for valuable assistance in working out the statistics of my cases.

138, *Harley Street,*
London, W.
May, 1903.

PREFACE TO THE SECOND EDITION.

I TAKE this opportunity to thank my *confrères*, both at home and abroad, for the very generous reception accorded to the work.

In preparing the second edition, nothing has been altered or omitted. But some small additions have been made, and some matters more fully discussed.

Dr. E. H. Oppenheimer has translated the book into German (for Messrs. Julius Springer and Co., Berlin).

PREFACE TO THE THIRD EDITION.

THE third edition contains statistics which give a fair idea of the results which may be expected from the methods of advancement and fusion training described in these pages.

May, 1906.

SQUINT:

Its Causes, Pathology and Treatment.

CHAPTER I.

INTRODUCTION.

THIS chapter contains nothing new. It deals very briefly with certain elementary facts.

EMMETROPIA is the refractive condition of the normal adult human eye. Rays of light proceeding from a single point on a distant object may, for practical purposes, be regarded as parallel. When these parallel rays enter an emmetropic eye, they undergo refraction as they pass through the refracting media (cornea, aqueous humour, lens, vitreous humour), and are brought to a focus on the retina. Rays from every other point in the distant object are similarly focussed, so that a complete (inverted) image of the object is formed on the retina. The refraction which takes place under these conditions is called the *static refraction* of the eye.

ACCOMMODATION.—Rays of light which enter the eye from a near object, *e.g.*, a printed page, are sensibly divergent. Now, it is obvious that the *static* refraction of the normal eye, which exactly suffices to bring parallel rays to a focus on the retina, will not accurately focus these divergent rays. To meet this

deficiency, there is a muscle within the eyeball, the ciliary muscle, which, by its contraction, causes the lens to become more convex, more nearly spherical, and so increases its refractive power. This act of increasing the refractive power of the eye is called *accommodation*, and the additional refraction thus produced is called the *dynamic refraction* of the eye.

PRESBYOPIA.—In childhood the lens is very soft and elastic, and is easily made to change its shape under the action of the ciliary muscle, so that children have a very wide range of accommodation. As age advances, the lens gradually becomes more and more firm and incompressible, so that, though distant objects are still perfectly focussed by means of the static refraction of the eye, the increase of refraction, produced by the action of the ciliary muscle on the lens, becomes, after a time, insufficient for the focussing of near objects. For this reason, a normal-sighted person of fifty must either hold his book at a greater distance, in order that the rays of light proceeding therefrom shall be more nearly parallel, or he must supplement his weakened accommodation with a pair of convex glasses.

ATROPINE, when instilled into the conjunctival sac, has the property of temporarily paralysing the ciliary muscle, and so suspending entirely the power of accommodation. An atropised normal eye sees distant objects distinctly, by virtue of its static refraction, but is quite unable to focus the divergent rays proceeding from a near object. In other words, atropine produces an artificial presbyopia.

FIXATION.—In the centre of the retina is the macula lutea, which, in the human eye, is far more sensitive to ordinary visual impressions than any other part. It is desirable, therefore, that the eye be brought into such a position that the image of any object which especially engages our attention shall be formed upon the macula lutea. The eye is then said to "fix" the

object. An imaginary line, passing from the centre of the macula, through the optical centre of the eye, to the object looked at, is called the *visual axis*.

CONVERGENCE.—When the two eyes look at a distant object, the visual axes may, for practical purposes, be considered to be parallel. When, however, a near object is looked at, the two eyes must rotate inwards, in order that both visual axes may be directed to the same object. This active inward rotation of the eyes is called *dynamic convergence*. In the case of a normal pair of eyes there is no such thing as *static convergence*, because the primary position of the visual axes is one of parallelism. In a case of convergent squint, however, there is a static convergence corresponding to the angle of the deviation.

ACCOMMODATION AND CONVERGENCE. — When a person with a normal pair of emmetropic eyes looks at a near object, the eyes converge in order that both visual axes may be directed to the object. At the same time each eye "accommodates," in order that the rays of light from the object may be accurately focussed on its retina. These two functions, accommodation and convergence, are, in ordinary life, always used together, so that they have become "associated" by hereditary and individual habit. It is difficult, therefore, for a normal pair of eyes to accommodate without converging or to converge without accommodating.

CONJUGATE MOVEMENTS.—In looking to the right, or left, or up, or down, the two eyes move together through exactly the same angle.

MOVEMENTS OF EACH EYE SEPARATELY. — The extreme range of upward and downward rotations of a single eye varies slightly in different people, the average being about 46° up and 56° down.[1] Outward

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[1] These figures are the average of measurements which I made on 64 normal-sighted persons with Stephens' tropometer.

rotation (abversion) may be considered full when the edge of the cornea can be made to touch the outer canthus. The power of inward rotation (adversion) varies considerably in different people. Most people can advert each eye separately through an arc of 50°. The power of independent adversion tends to become less as age advances.

Hypermetropia.—For purposes of discussion, the refracting media of the eye may be diagrammatically represented as a simple convex lens. In the emmetropic eye, as already explained, the strength of this lens is such that parallel rays of light are brought to a focus exactly at the retina. A hypermetropic eye is shorter, from before backwards, than the emmetropic eye. This abnormal shortness causes the retina to be situated too near this diagrammatic lens. Now, in order that parallel rays may be brought to a focus on this abnormally situated retina, the focal length of the diagrammatic lens must be shortened, or, in other words, the strength of the lens must be increased. This increase in strength may be brought about either by the patient's using his accommodation in distant vision (and still more, of course, in near vision) or by his wearing a convex spectacle lens in front of the eye. A hypermetrope may be able easily to accommodate sufficiently to correct his refractive error in distant vision, but may have difficulty in sustaining the additional effort of accommodation involved in looking at a near object, *e.g.*, in reading.

Myopia is the converse of hypermetropia. A myopic eye is abnormally long from before backwards, so that the retina is at a greater distance from the centre of our diagrammatic lens than is the case in the emmetropic eye. In order, therefore, that parallel rays from a distant object shall be focussed on the abnormally situated retina, the focal length of the diagrammatic lens must be increased, *i.e.*, the strength of the lens must be diminished. The only way in which this

can be accomplished is by putting a concave spectacle lens in front of the eye.

If an indistinctly seen distant object be gradually brought nearer a myopic eye, the rays which enter the eye therefrom become more and more divergent, till a point is reached at which the static refraction of the myopic eye is just sufficient to focus these divergent rays. If the object be brought still nearer, the eye accommodates and so still sees distinctly.

ASTIGMATISM.—In the human eye, the greater part of the refraction takes place at the surface of the cornea, where the rays of light pass from the air into the much denser medium, the corneal substance. The normal human cornea is approximately a segment of a sphere. Sometimes, however, the cornea is curved more in one meridian than in another, so that it is slightly oval—like a slice from the side of a cocoa-nut. This condition is called astigmatism. In the meridian of greater curvature the rays will be refracted more, and brought to a focus sooner, than in the meridian of less curvature. Consequently the picture formed on the retina will be blurred and indistinct. In order to equalise the refraction in the different meridians, a cylindrical lens must be used, *i.e.*, a lens which is curved in one direction only.

ANISOMETROPIA is an inequality in the static refraction of the two eyes.

ANGLE GAMMA.[1]—The visual axis does not exactly correspond with the geometrical anterio-posterior axis

[1] The angle gamma which I have described is the angle which is measured clinically and is of practical importance. But, since the visual axis need not necessarily pass through the centre of motion of the eye-ball, mathematicians have taken as one of the boundaries of the angle gamma a line passing through the centre of motion of the eye-ball to the object looked at. This line is of no clinical significance as its direction cannot be determined except on paper.

of the eye-ball. The angle between the visual axis and the antero-posterior axis of the eye-ball is called the *angle gamma*. Usually the visual axis passes through the pupil to the nasal side of its centre. Rarely it passes through the pupil to the temporal side of its centre, in which case the angle gamma is said to be negative.

In hypermetropic eyes the angle gamma is usually high, so that, when the visual axes are parallel, the antero-posterior axes of the eye-balls are perceptibly divergent. In this way a deceptive appearance of divergent squint may be produced or a slight convergent squint may be masked.

In myopic eyes the angle gamma usually is low or even negative. In the latter case, while the visual axes are parallel, the antero-posterior axes of the eye-balls are convergent, so that convergent squint may be simulated or a slight divergent squint masked.

CHAPTER II.

BINOCULAR VISION.

When the eyes are in the primary position (*i.e.*, looking straight ahead into the distance) the fields of vision of the two eyes overlap everywhere, except in a sector of about 35° towards the temporal periphery of each field. In other words, everything which a normal-sighted person sees, he sees with both eyes simultaneously, except objects which lie on his extreme right or his extreme left. These are seen only with one eye.

When a distant object engages our attention, the two eyes are brought into such a position that a picture of the object is formed simultaneously on the central part of each retina. Similarly, all other distant objects, within the limits of overlapping of the visual fields, are focussed on functionally corresponding parts of each retina. The impressions thus received from the two eyes are blended in the brain, so that we are conscious only of one single picture. This psychical blending of the two sets of visual impressions is called binocular vision.

The binocular vision of near objects is a more

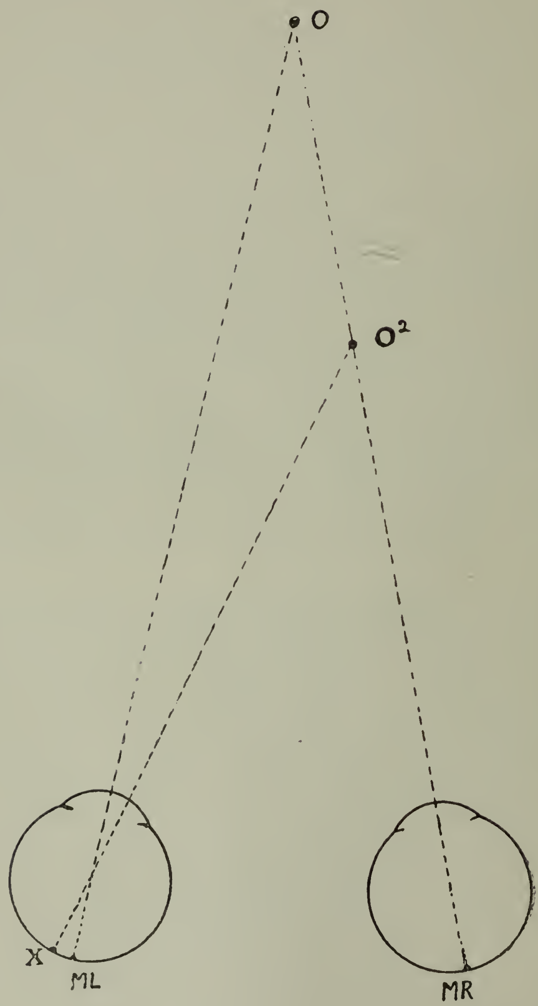

FIG. 1.

complex act, inasmuch as the brain has to blend images which, for the most part, do not fall upon geometrically corresponding points of the two retinæ.

The diagram represents a pair of eyes looking at an object O in the middle distance. An image of O is formed on the macula ML and MR of each retina. Now, it is obvious that only objects lying in the line MR—O (or in this line produced beyond O) can be focussed on the macula of the right eye. Similarly, only objects lying in the line ML—O can be focussed on the macula of the left eye. O^2 is an object lying in the line MR—O. An image of O^2 will be formed on the macula of the right eye. But the image of O^2 formed in the left eye will not be on the corresponding point, but at a point X, considerably to the outer side of the macula.

Take a practical illustration—look steadily at a distant object, and hold a finger about 18 inches in front of the eyes. The finger will be seen double, the left image corresponding to the right eye, and the right image to the left eye (crossed diplopia). Now look at the finger, and the distant object will appear double, the diplopia this time being homonymous.

This "physiological diplopia" must be constantly present, in looking about a room for example, yet we are not ordinarily conscious of seeing double. This customary freedom from

diplopia is brought about, not by mental suppression of one of the images, but by the marvellous elasticity of the fusion faculty. Both sets of impressions reach the brain, and, by their combination, assist in our appreciation of the third dimension.

A simple experiment will demonstrate the elasticity of fusion. Place in the amblyoscope (chapter viii.) the slides shown in fig. 13, p. 124. The two slits are fused into one, and the control marks are seen, one on each side. Now gradually diverge the tubes. When the extreme limit of divergence of the visual axes has been nearly reached, fusion of the slits is still maintained, but the control marks recede on each side farther and farther from them. When the limit is passed and fusion can no longer be maintained, the slits suddenly spring apart. As each slit is on the same slide with its control mark, the distance cannot really vary ; but, within certain limits, the mind still fuses the images of the slits, even when they no longer fall upon anatomically corresponding points of each retina. This experiment would serve as a proof, were any needed, that fusion is a purely psychical process, and not merely the result of stimulation of corresponding sets of nerve endings in each retina.

But this must not be taken to indicate that accurate adjustment of the relative positions of the two eyes is unnecessary. The *law* governing

the fusion of images which are not precisely similar may be stated as follows: *When the images formed in the two eyes differ in shape, size, or position, if the disparity be not too great, the oculo-motor apparatus first places the eyes in the most favourable relative positions; the fusion sense, by virtue of its elasticity, then fills up any gap which may remain.*

The following additional experiments were suggested to me by Dr. Verhoeff, of Boston, U.S.A.

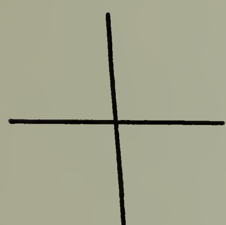

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

Place fig. 2 and fig. 3 in a stereoscope. The horizontal lines of course are blended. But the oblique lines also are blended; the resulting line appearing perpendicular to the horizontal line. This

blending of the oblique lines cannot be accomplished by any rotation of the eyeballs round a fore and aft axis, as, in this case, the horizontal lines would not be blended.

Again, if figs. 4 and 5 be blended in a stereoscope the combined image resembles fig. 6. The oblique lines outside the circles are blended into a perpendicular line, but the part within the circle, which is seen with one eye only, retains its oblique direction.

In a case in which each eye separately has the power of seeing, but in which binocular vision is absent, one of two conditions must be present, either (*a*) the mind is separately conscious of the two sets of impressions received from the two eyes — diplopia, *e.g.*, in paralysis of an external ocular muscle; or (*b*) the mind takes note only of the impressions received from one eye and ignores those received from the other —suppression, *e.g.*, in an ordinary case of convergent squint.

Grades of Binocular Vision.

Most people who have binocular vision have the faculty to its full extent. Anyone, however, who has undertaken the orthoptic training of any considerable number of squinters, will find that those who see binocularly naturally arrange themselves into three separate and distinct classes, according to the degree in which they possess the faculty.

These may be called :—

First Grade.—Simultaneous macular perception.

Second Grade.—True fusion with some amplitude.

Third Grade.—Sense of perspective.

First Grade. Simultaneous macular perception. — A patient having only this grade of binocular vision sees devices in a stereoscope as two separate pictures, which overlap and form one only when they are put in certain relative positions corresponding to the directions independently assumed by the visual axes. The "desire" for binocular vision is absent, so that no effort will be made to maintain fusion.

Second Grade. True fusion with some amplitude.—A person having the second grade of binocular vision not only fuses the retinal images in the two eyes, but can make some effort to maintain fusion. When such a person is fusing the pictures in a stereoscope, if the pictures be separated or brought together the eyes will, to a certain extent, follow them in the interest of binocular vision.

Third Grade. Sense of perspective.—The two eyes see from different points of view. In looking at any solid object, such as a pillar, for instance, the right eye will see more of the right side of the object and the left eye more of the left side. In the slightly dissimilar pictures thus focussed on the retinæ the points of difference are not suppressed, as in the case of a person having

only the second grade, neither is the observer conscious of diplopia. The psychical blending of the two slightly dissimilar sets of visual impressions enables him to appreciate the solidity of surrounding objects and assists in his judgment of their relative distances.

There is a wide gap between grades 1 and 2. A patient, however, who has grade 2 usually acquires a third grade also.

Quite as important as the grade of binocular vision is its intensity. A person whose fusion sense is feebly developed may possibly, under favourable conditions, have the highest grade of binocular vision. But the intensity of his tendency to fusion will be slight, so that, under unfavourable conditions, he easily abandons the effort and uses one eye only. One, however, whose fusion sense is well developed will have such an intense tendency to binocular vision that nothing will make him abandon it while both eyes are open. (Except, of course, a muscular paralysis, in which case he will suffer from persistent and intolerable diplopia.)

Tests for Binocular Vision.

Four dot test. — A convenient clinical test, which I have used constantly for some years, is an adaptation of Snellen's coloured glasses. For want of a better name, it may be called the "Four dot test." A pure red glass allows

only the red rays of light to pass through it. A pure green glass transmits only the green rays. Therefore light which has passed through

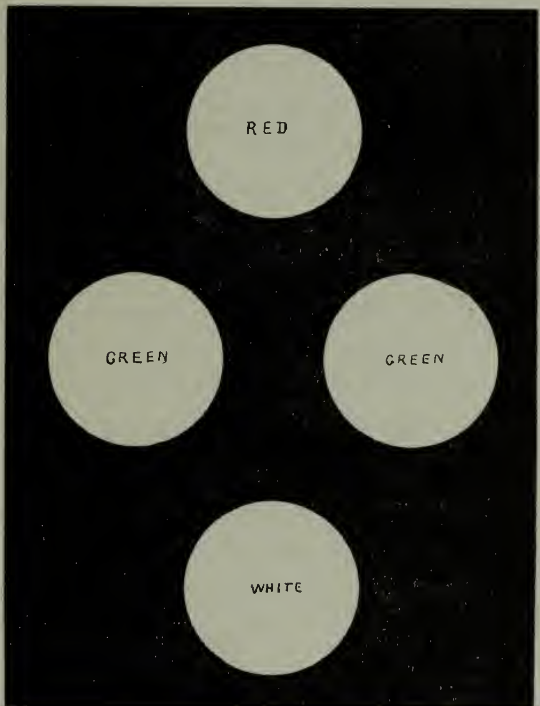

FIG. 7.

the red glass cannot be seen at all through the green glass, and *vice versa.*

A piece of plain ground glass, 12 inches by 9 inches, is covered on the back with opaque black paper. The black paper has four round holes cut in it, each 3 inches in diameter, as shown in the

diagram. The lower hole is left clear. Behind the upper hole is cemented a piece of red glass. Behind each of the other two is cemented a piece of green glass. The arrangement is mounted in the front of a box which contains an electric or other bright light.

The patient, standing five or six yards away, wears a trial frame with a red glass before the right eye and a green glass before the left. If now he sees two dots (white and red) he is using the right eye only. If he sees three dots (white and two green) he is using the left eye only. If he sees four dots (white, red, and two green) he uses both eyes, and has at least grade 1 binocular vision. If he sees five dots (red, two green, and the white seen double) he has diplopia. If the accuracy of the patient's answers be doubted, it may be tested by changing the glasses in the spectacle frame from one eye to the other.

The amblyoscope (see chapter viii.).—Adjust the instrument for parallelism of the visual axes. Place in the slots the slides shown in fig. 13. If the patient sees the two slits as one, and, at the same time, sees both the dot and the cross, he has grade 1 binocular vision. Now diverge or converge the tubes of the instrument. If this can be done, even to a very slight extent, while the patient still fuses the slits and sees both control marks, he has grade 2 binocular vision.

For children, more interesting objects, such as figs. 16 and 17, may be used in the same way. The extent to which the tubes may be separated or brought together without the eyes becoming dissociated, may, for practical purposes, be taken as a measure of the degree of development of the fusion faculty.

Now put in the instrument the slides shown in fig. 18. A patient with grade 2 binocular vision will only fuse the outer circles; but he will either suppress the image of one of the inner circles, or will see both inner circles "all mixed up." A person, however, with grade 3 binocular vision sees the inner circles blended and appearing much nearer the eye, giving the whole device the appearance of a tub or bucket bottom up. If the slides be now changed from one tube to the other, the inner circle will seem farther away, as if he were looking at the inside of the tub. This appearance is so vivid that even young children can tell at once whether they are looking at the outside or the inside of the tub. By changing the slides from one tube to the other two or three times, guessing on the part of the patient is rendered impossible. I know no better test for the "sense of perspective."

Hering's drop test for the sense of perspective.—I do not now use this test clinically. The experiment is, however, very instructive.

The test apparatus (fig. 8) consists of a shallow box (of about the size and shape of a box for twenty-five large cigars), open at both ends. From one end two arms project. The extremities of these two arms are joined by a fine thread, on the middle of which is a round bead.

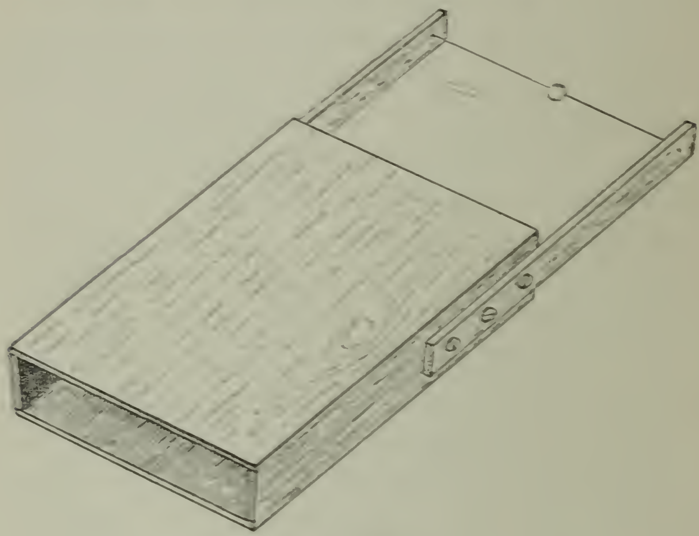

FIG. 8

The patient holds the open end of the box close to his eyes, and looks through the box, at the bead. The surgeon now drops small objects of various sizes, such as cowrie shells, sometimes on one side of the thread, sometimes on the other. The patient is asked to say whether each shell, as it drops, falls on the near side or the far side of the thread.

If he has the third grade of binocular vision, "the sense of perspective," he will almost always answer correctly. If he has not, his answers will be mere guesses, and he will be nearly as often wrong as right.

The principle of the test is as follows:—The box cuts off all view of surrounding objects, including the hands of the surgeon. The size of the falling objects varies, so that their apparent size gives no information as to their distance. The view of the falling object is too brief to admit of any movement of accommodation or convergence, or any lateral movement of the patient's head. The patient is thus deprived of all auxiliary means of judging distances, and has to depend upon his "sense of perspective" alone.

This test is not infallible. I once made the discovery that a boy, aged 11, with a manifest squint of 13°, could tell on which side the shell fell, almost every time, when both eyes were open. When, however, I covered the deviating eye his answers were as often wrong as right. He had, in the deviating eye, $\frac{6}{18}$ vision. There was no "false macula." He had no diplopia spontaneously, but it was easily elicited with a candle and coloured glasses. I have since met with other similar cases. The explanation is probably this:—In trying the test with a normal pair of eyes, the view of the falling shell is so brief that there is no time for the eyes "to fix" the shell, so the images of the object do not fall upon corresponding parts of the two retinæ. It is not, therefore, really a test for the sense of perspective in direct vision, but a test for

the subconscious perception of "physiological diplopia" in more or less eccentric parts of the retinæ.

In none of these cases was the suppression of the vision of the deviating eye very profound, so that a moving object would probably be perceived. As strictly corresponding points are not required in this test, it seems not unlikely that the mind may derive some information from the false image, by making allowance for the faulty position of the deviating eye. This is not difficult to believe when we remember that, in a case of squint in which diplopia has been artificially elicited, the angle of the diplopia is often very much smaller than that of the squint; showing that the mind ordinarily makes some allowance for the position of the squinting eye.

The Normal Development of the Fusion Sense.

In two large crêches I made experiments, extending over nearly a year, with the object of gaining some knowledge of this subject. It would be tedious to describe in detail the methods employed. I therefore give a general summary of results.

From the earliest infancy the pupillary light reflex and the fixation reflex are present, showing that some degree of vision of each eye, and the preponderance of the macula region, are innate. If, in a darkened room, the light of a candle be suddenly thrown from an ophthalmoscope mirror into an eye of an infant only a few hours old, the eye will immediately fix the mirror. This fixation is purely reflex and is only maintained

for an instant. During the first few days of life the infant cannot fix a steady light, but, by suddenly flashing the light into the eye, this reflex fixation may be repeatedly obtained.

At the end of two or three weeks most infants will fix the mirror steadily for several seconds at a time with one or other eye, but will not converge both visual axes accurately in looking at a near object.

At the age of five or six weeks, as a rule, the positions of the reflections of the mirror on the child's corneæ are symmetrical, showing that the child is fixing the mirror binocularly. But, now and then, one eye turns a little inwards, or more rarely outwards, while the other fixes the mirror.

During the first few months of life the movements of the eyes are uncertain, not completely controlled by the higher centres of the brain. The eyes move more or less together, but the slightest gastric or other disturbance often causes one or other eye to deviate. But it will be noticed that this want of coördination is confined to movements in the horizontal plane. The conjugation of the two eyes for vertical movements is well-developed from the earliest infancy: one does not see one eye turn up or down without the other.

We can scarcely suppose that any degree of binocular vision can be present at a time when

the coördination of the eyes in horizontal movements is in this rudimentary condition.

A little later, at the age of five or six months, if the child's attention be engaged by some new and bright object of absorbing interest, such as a cut glass decanter - stopper revolved before a candle flame, it will often be possible for the person nursing the child to slip a large prism before one of his eyes without his appearing to notice it. A prism displaces the image of an object towards its apex. Therefore, if binocular vision is to be maintained, the eye must also rotate in the direction of the apex of the prism. If a prism of, say, 12° has been successfully slipped before one of the child's eyes, with the apex towards the nose, the eye will, in many cases, make a slight inward rotation, showing that the child has some sort of binocular vision. In some cases, however, while the naked eye continues to be steadily directed towards the object, the eye behind the prism makes no inward rotation. The vision of this eye is probably suppressed. These resemble certain cases of occasional squint, in which binocular vision is present when the eyes are "straight," but suppression of the vision of the deviating eye takes place when the squint is manifest.

After the end of the first year, a child who allows one to make the experiment with the

prism apex inwards will almost always turn in the eye in order to blend the images.

When the experiment is tried with the prism apex up or down, it is, of course, not possible for the eye to make an independent vertical movement in order to blend the images. I tried this vertical prism with some of the more tractable infants, aged twelve to eighteen months, who had readily made the compensating inward movement when tried with the prism apex in. Some of them showed their disapproval of the vertical prism by screwing up the eyes and twisting the head. Others made up-and-down conjugate movements of both eyes, sometimes directing their attention to the upper image and sometimes to the lower.

To recapitulate — The vision of each eye separately, the preponderance of the macular region, and the conjugation of the two eyes in vertical movements, the human infant has fairly well developed at birth. The conjugation of the eyes for horizontal movements (intended to subserve the function of binocular vision) is perfected within the first few months of life. Between five and six months one finds the first certain evidence of a "desire for binocular vision," though probably a certain degree of binocular vision is present at a much earlier period. At first, if any obstacle be interposed, it is a question whether an effort shall be made to overcome it, or whether the newly acquired art

shall be abandoned and the vision of one eye temporarily suppressed. Towards the end of the first year the eyes will make a considerable effort in the interests of binocular vision. If the obstacle prove insuperable the child suffers from diplopia, being no longer able to suppress the vision of one eye.

The results of fusion training in the case of squinters would seem to show that the fusion faculty normally reaches its full development before the end of the sixth year.

CHAPTER III.

CONVERGENT SQUINT.

In most of the text-books squint is defined somewhat as follows : " Squint consists in a deviation of the visual axis of one of the eyes from the correct position of fixation." The authors have mistaken a single symptom for the whole disease. One might as well describe Pott's disease as " a deviation of the spine from its normal shape."

Two essential conditions are present in every case of comitant convergent squint.

(1) An abnormal convergence of the visual axes.

(2) A defect of the fusion faculty.

Other conditions may also be found :—

(3) The vision of the eye which is not being used for fixation is almost invariably suppressed.

(4) There is, in rather rare instances, more or less congenital amblyopia.

(5) There is very often acquired amblyopia in the deviating eye, as the result of neglect or inefficient treatment.

(6) There is usually a refractive error, com-

monly hypermetropia and hypermetropic astigmatism.

In a healthy person, with a normal fusion faculty, the "desire for binocular vision" causes the two eyes to be directed steadily to the same object. But when the "desire for binocular vision" is absent there is no special reason for this perfect accord between the movements of the two eyes, so that any slight cause may then upset the equilibrium of the convergence centre, and so cause the visual axes to assume permanently faulty relative directions (see chapter iv.).

Then, when the patient is looking at a distant object, instead of the visual axes being parallel they are convergent. But, in order to see the object distinctly, the patient must look directly at it with one or other eye. He will naturally choose the eye which has the smaller refractive error. He cannot overcome the abnormal convergence, neither can he move one eye without the other. He therefore makes a conjugate lateral movement of both eyes until he has brought the better eye into the required position, the other eye turning still more in towards the nose. So that the better eye becomes "straight," and the worse eye manifests the convergence of both. The eye which is used for vision is called the fixing eye ; the other eye is called the squinting or deviating eye.

If a patient fixes, say, with the right eye and

turns in the left, he is said to have convergent squint of the left eye. This is a useful convention, but it must be remembered that it does not accurately describe the condition, as, of course, convergent squint really concerns both eyes and certain cerebral functions as well.

In a case of convergent squint the separate movements of each eye are perfect. When one eye is covered, the other eye can move upwards (superversion), downwards (subversion), inwards (adversion), and nearly always outwards[1] (abversion) to the normal extent.

The conjugate movements of the two eyes are perfect. When the fixing eye moves in any direction, the deviating eye also moves through exactly the same angle. When the (previously) fixing eye is screened, and the patient fixes with the (previously) squinting eye, the screened eye manifests a deviation exactly equal to that formerly exhibited by the other eye.[2] In other words, the squint is comitant.

The association between accommodation (dy-

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[1] In 1,523 cases of convergent squint, in which I noted the power of abversion of each eye separately, I found it perfect in 81 per cent. The remaining 19 per cent., in which abversion was deficient, were almost all cases of long standing. The defect of abversion was less uncommon the longer the duration of the deviation.

[2] In a case of uncorrected anisometropia the patient may accommodate to a different degree, according to the eye he uses. This may make the squint appear to be not strictly comitant.

namic refraction) and dynamic convergence is perfect. When the fixing eye, after looking at a distant object, suddenly accommodates for a near object, the squinting eye rolls still further inwards, a dynamic convergence being superadded to the abnormal static convergence. This dynamic convergence is proportionate to the extra effort of accommodation involved in looking at the near object.

In fact, there is no motor defect of any kind in a typical case of convergent squint, but the primary position from which these movements start is a "cross-eyed" position instead of parallelism of the visual axes.

Convergent squint presents certain clinical varieties. These may be classified as follows:—

(1) Occasional squint, of which there are two classes: (*a*) Premonitory occasional. (*b*) True occasional.

(2) Constant unilateral squint.

(3) Alternating squint, of which there are two distinct classes: (*a*) Accidentally alternating squint. (*b*) Essentially alternating squint.

(1) *Occasional squint.*—A patient is said to have an occasional squint if the eyes are only occasionally seen to deviate from their normal relative directions. When no deviation is present, the patient usually has the first grade of binocular vision. When he squints he, in the majority of cases, has no diplopia. The deviation of an occa-

sional squint is in some cases only seen for a few seconds in the day, in others the eyes are almost as often "crossed" as not. The deviation in different cases may manifest itself under various conditions, *e.g.*, in near vision, on looking down, under the influence of any strong emotion such as fear or anger, when the patient is tired, &c. Most frequently no immediate exciting cause can be assigned. Occasional squints are sometimes called *periodic*. I have notes of a few cases which may properly be called periodic squints, in which the deviation has appeared every alternate day as regularly as a tertian ague.

(*a*) Premonitory occasional squints are simply the precursors of constant squints. They usually become constant after about two or three months.

(*b*) True occasional squints are much less common than the premonitory variety. A true occasional squint may gradually cease to show itself as the fusion sense develops, or perhaps optical correction of any refractive error may relieve the condition, or it may maintain its character unchanged throughout life. Many of these last are not squints at all, in the strict sense of the word, but are examples of esophoria, the pathology and treatment of which are described in chapter xi.

(2) In a case of *constant unilateral* squint the deviation is constantly present, though the angle may vary; and it is always manifested by the

same eye, when both eyes are uncovered. When the "fixing eye" is screened, it turns in towards the nose, and the "squinting eye," instead, is directed to the object which engages the attention (unless this eye has lost the power of central fixation). When the screen is removed, the fixing eye immediately recovers itself, and the squinting eye again turns in.

(3) *Alternating squint.*—When a squinter fixes with either eye indifferently, without covering the other, the squint is said to alternate. Of all constant squints, about 85 per cent. are unilateral and 15 per cent. are alternating. These alternating cases arrange themselves into two distinct classes—(*a*) Squints which accidentally alternate, because the refraction is about the same in each eye, do not essentially differ from unilateral squints. In fact, if these cases are untreated, usually one eye gradually comes to be used exclusively for fixation, and the squint becomes unilateral.

(*b*) In a case of essentially alternating squint there is usually little or no refractive error, and the visual acuity of each eye separately is, as a rule, perfect. But these patients have a congenital total inability to acquire fusion. Alternating squinters suffer much less than unilateral squinters from neglect, because each eye is used in turn, so that the vision does not deteriorate. If the wearing of glasses does not cause the

abnormal convergence to disappear (it seldom does), an accurately performed operation will remove the deformity. But the total absence of the fusion sense renders a perfect cure of an essentially alternating squint impossible.

Suppression of the vision of the deviating eye.—In a case of convergent squint, as the two eyes are not directed towards the same object, it might be thought that everything would be seen double. This is not so, however, save in the exceptional instances referred to below. The visual acuity of the deviating eye may be perfect, but the picture formed in this eye is mentally ignored or "suppressed," the attention being directed solely to that formed in the fixing eye. This "suppression" is not a voluntary act. The inability to receive impressions from both eyes simultaneously is due to the defect of the fusion faculty, which was the essential factor in allowing the squint to occur in the first instance.

This suppression, however, does not always extend over the whole field of vision of the squinting eye. If the deviation is of only slight degree, there is a small part of the temporal side of the field of vision of the squinting eye which lies beyond the limit of the field of the fixing eye. In this small area moving objects are perceived by the squinting eye, but, as a rule, are not accurately located. Thus, in an old case of convergent squint, in which the deviating eye

has lost the power of central fixation, the extreme nasal side of the retina of this eye (corresponding to the extreme temporal side of its field of vision) is the only part which is ever exercised. For this reason, if the fixing eye in such a case be covered, and the patient be told to look at a light with the deviating eye, this eye will be seen to roll still further inwards, in order to receive the image of the light on the nasal side of the periphery of the retina.

Occasionally one meets with a squinter who has diplopia of a faint, unobtrusive kind. Squinters never complain of diplopia as a trouble, but one now and then meets with a child who, on being carefully questioned, admits seeing a second image. If the white handle of an ophthalmoscope be held up, such a child will point to the real handle and also to "the sham one," showing that he really has a faint homonymous diplopia, though, in the great majority of cases, he has never mentioned it before. In such a case he evidently has some trace of a fusion faculty, but not sufficiently well developed to prevent the occurrence of a deviation.

Diplopia artificially produced.—In a case of unilateral or accidentally alternating squint, if the deviating eye be not too blind, diplopia may usually be induced by artificial means. For instance, let the patient's attention be directed to a candle flame. Place in a spectacle frame

a red glass before one eye and a green glass before the other. The images of the candle flame, formed in the two eyes, being thus differently coloured, the patient is often enabled to perceive them both simultaneously.

Nature of diplopia.—The popular idea is that a squinter sees the object which lies in the axis of the deviating eye as well as that which lies in the axis of the "straight" eye, so that he can keep an eye on two places at once. Even in the case of the very few squinters who are able to see double spontaneously, or of those who are enabled to do so by artificial means, this view is not correct. A squinter who suffers from diplopia sees, with his deviating eye, a faint, eccentrically-placed image of the object to which the fixing eye is directed, and suppresses the image of the object which lies in the axis of the deviating eye. In other words, he does not see two different objects, but sees two images of the same object.

The direction in space in which this second image is mentally projected is peculiar.

Now, in a case of paralysis of, say, the right external rectus muscle, if the right eye deviates inwards to the extent of 20°, everything seen with this eye will appear to be exactly 20° more to the right than it really is ; in other words, there is homonymous diplopia of 20°. If an eye turns out to the extent of 20° owing to paralysis, there is crossed diplopia of exactly 20°. The

mind entirely ignores the faulty position of the eye, and projects everything rigidly as though the eye were straight. In a case of convergent squint, on the other hand, if there is diplopia this does not necessarily correspond in degree with the angle of the deviation of the eye. The eccentrically-formed image is, in any case, very faint. Even the most intelligent patient usually is unable to describe its position exactly, as the angle of the diplopia seems to vary without any corresponding variation in the angle of the squint. In most cases the false image is placed about half-way between the true position of the object and the position which would correspond to the angle of the squint. It would seem as though the mind, being informed by the straight eye of the true position of the object, were continually trying to reconcile this knowledge with the impressions produced by the eccentrically-placed false image.

The amblyopia of convergent squint.—When a patient first comes under observation after having suffered from unilateral squint for a considerable time, one usually finds that the deviating eye is more or less blind, so blind sometimes that fingers can scarcely be counted close to the face. This amblyopia is sometimes, to a certain extent, congenital. But by far the greater part of it is due to a gradual loss of function in an eye which is never used. It might have been prevented.

This is plainly seen by comparing the vision in my cases which came under treatment soon after the first appearance of the squint, with the vision in the cases which I saw for the first time only after years of neglect or inefficient treatment (see pages 76 and 77).

The power of central fixation in the deviating eye.—In a case of unilateral convergent squint, if the fixing eye be covered the vision of the (previously) deviating eye temporarily ceases to be suppressed. In a fairly recent case, this eye is then directed so as to receive, upon its macula lutea, the image of the object looked at. But if the case be long neglected, this sensitive central region of the retina suffers much more from disuse than the paracentral zone, while the peripheral region suffers very little, if at all. As the blindness progresses in this disused eye, a stage is at length reached when the visual acuity of the central region falls below that of the paracentral zone, and later, even below that of the periphery of the retina. If the fixing eye be now covered, the deviating eye is not directed so as to receive upon its macula the image of the object which engages the attention, because the macula has ceased to be the most sensitive part of the retina. This eye then wanders, without remaining steadily in any definite position (*lost fixation*). Or it may fix with some part of the paracentral region, or roll still further in towards the nose so as to

present the extreme nasal periphery of the retina for the purpose (*false fixation*).

False macula.—False fixation is, unfortunately, exceedingly common in neglected cases of unilateral squint. But the variety known as false macula is rare. In an old case of squint, in which the angle of the deviation has remained exactly the same for several years, and in which the suppression of the vision of the deviating eye is not profound, the mind sometimes learns to make full allowance for the faulty position of this eye. So that the eccentric image, formed in the deviating eye, is mentally projected to the same spot as the true macular image, formed in the normally-directed eye, and is blended with it. This false macula is merely a small area which has escaped the loss of function which has overtaken the surrounding part of the retina. The visual acuity of a false macula is never greater than the normal visual acuity of the region in which it is situated. I have rarely found it equal to $\frac{6}{60}$; never greater. Many of these patients can pass Hering's drop test. If an eye, with false macula in a position of convergence, be put "straight" by operation, crossed diplopia is produced. This usually passes off within a few days, but occasionally it persists for many months.

Monocula diplopia.—In an eye with "false macula," central fixation has nearly always been lost. Occasionally it is preserved. In such a

case, if the fixing eye be covered, the deviating eye will immediately take up central fixation. The "false macula" is then, as a rule, suppressed. If it is not, the result is monocular diplopia. This condition is exceedingly rare. In my detailed notes of more than two thousand cases of squint of all kinds, I find monocular diplopia only mentioned four times, although I have been constantly looking out for this anomaly.

Apparent vertical deviation.—On applying the mirror test in a case of convergent squint, one not infrequently notices a slight upward deviation also: then, on covering the originally fixing eye, and causing the originally deviating eye to fix, one sees that the originally fixing eye also turns *up* as well as in. If the wearing of glasses causes the convergence to diminish, the vertical deviation will also diminish proportionately. In the great majority of cases the vertical deviation increases when the gaze is directed towards the side of the fixing eye and diminishes or disappears when the gaze is directed away from the side of the fixing eye.

In some cases of divergent squint, the divergent eye turns a little down also, no matter which eye may be fixing.

A possible explanation is that in these cases the planes of adversion and abversion of the two eyes, instead of being horizontal, are titled down and out towards each temple. Then, when the fixing eye is

moved horizontally, the deviating eye, in making a corresponding conjugate movement uncontrolled by the fusion sense, moves in this titled plane. Take, for example, a case of convergent squint 20°, exhibiting this double upward deviation. Really each eye is convergent 10°, but the eyes have made a conjugate movement of 10° towards the side of the fixing eye so as to bring this eye " straight " and double the apparent convergence of the deviating eye. The fixing eye has thus travelled out and down in its plane, and the deviating eye has travelled in and up. A further conjugate movement towards the side of the fixing eye will increase the difference in vertical height. A conjugate movement away from the side of the fixing eye, by bringing the eyes to corresponding points in their respective planes of rotation, will cause the vertical difference to lessen or disappear.

True vertical deviation.—In some cases of squint there is a true vertical deviation, one eye turning up when it becomes the deviating eye, and the other eye turning down when it, in turn, is made to deviate.

Spurious squint of infants.—During the first few months of life, before the fusion faculty has made much progress in development, it frequently happens that the eyes converge for a few seconds or a minute at a time, in response to some gastric or other disturbance, Or, as nurses are wont to express it, " Babies squint when they have the wind." This is of no importance. Sometimes, too, a child, whose fusion faculty is developing quite normally in other respects, acquires this faculty rather later than usual, just as a perfectly

healthy, intelligent child may be late in learning to talk. One may then see one or other eye turn in, occasionally, for a minute or two at a time, even when the child is old enough to walk. Later, if the fusion faculty developes normally, it will so control the movements of the eyes as to prevent any recurrence of the deviation.

But if the child "crosses his eyes" for many minutes at a time, or if one eye converges while the other steadily fixes some object, or, more especially, if the deviation is always manifested by the same eye, the case is probably one of true squint and demands investigation without delay. And, under any circumstances, it is safer to investigate any case in which a child is seen to cross his eyes occasionally, rather than wait until what may have been a premonitory occasional squint becomes a constant squint.

"*Growing out of a squint.*"—With the advent of puberty, the angle of a convergent squint often tends to become somewhat less without any treatment. In rather rare instances the eyes become straight, or nearly so, and the patient is said to have "grown out of the squint." But, in the case of a unilateral squint, the squinting eye has nearly always by this time become very blind from disuse.

The belief in the spontaneous cure of squint is very wide-spread among the general public. This may be due partly to the fact that the deformity

of squint does, in a few cases, disappear spontaneously as just mentioned, and partly to the fact that an occasional squint is seen at one time and not at another, but chiefly, I think, to the fact that the spurious squint of infants ceases to manifest itself when the fusion sense developes.

But, unfortunately, this superstition is not entirely confined to the laity. I have often had a child brought to me with the squinting eye nearly blind from neglect, and have been told that the family practitioner was consulted about it years ago and that he advised the parents to "wait to see if the child would grow out of the squint."

General course of an untreated case of constant unilateral convergent squint. — At the first appearance of a deviation, the squinting eye always has the power of central fixation when the fixing eye is covered, and the vision is nearly always good in both eyes. There is rarely some congenital amblyopia. This congenital amblyopia is far less frequent than is generally supposed, and is never responsible for the extreme blindness so often found in old neglected cases of squint.

In an untreated case, the vision of the deviating eye, being entirely suppressed, gradually deteriorates from disuse, until, in many cases, central fixation is lost, and the vision reduced to the counting of fingers close to the face. The younger the child the more readily does this

amblyopia from disuse occur. So much so that it is commonly believed, even by ophthalmic surgeons, that an eye which begins to squint in early infancy is necessarily very blind, and this blindness is supposed to be congenital. Yet this is not the case. Of the cases which came under my care soon after the first appearance of the deviation I do not find the young squinters especially amblyopic (see chapter v.).

The deviation, in an untreated case of convergent squint, usually increases in degree up to the time of puberty. After this time it often tends to become gradually less, until about forty years of age, when in some cases it is scarcely noticeable. It is not very uncommon to see a middle-aged patient with an inconspicuous squint of perhaps two or three degrees, and a nearly-blind eye. He usually says he squinted when he was a boy, but gradually grew out of it.

The fusion sense, of course, cannot be developed during the continuance of a deviation, except by artificial means (*e.g.*, exercises with the amblyoscope). If it be prevented from developing in infancy and early childhood, it will never develop at all to any useful extent. It is true that binocular vision of a sort may, in exceptional cases, be acquired as late as eight or nine years of age, but it is so feeble that it is powerless to maintain the normal relative direction of the eyes in the presence of uncorrected hypermetropia;

so that glasses must be worn throughout life to prevent a return of the deviation.

Age at which the deviation first appears.—In my note-books I find the onset-age recorded in 1,017 cases of unilateral convergent squint, and in 178 alternating cases.

The number of *unilateral* cases beginning in each year of life was as follows:—

Before 1 year	134	cases.
Between 1 and 2 years	186	,,
,, 2 ,, 3 ,, ...	247	,,
,, 3 ,, 4 ,, ...	189	,,
,, 4 ,, 5 ,, ...	113	,,
,, 5 ,, 6 ,, ...	73	,,
After 6 years	75	,,

It will be seen that in nearly 75 per cent. of the cases the deviation appeared before the end of the fourth year, and in less than 7½ per cent. its advent was delayed until after the sixth year.

The *alternating* cases work out as follows:—

Before 1 year	61	cases.
Between 1 and 2 years	34	,,
,, 2 ,, 3 ,, ...	23	,,
,, 3 ,, 4 ,, ...	29	,,
,, 4 ,, 5 ,, ...	11	,,
,, 5 ,, 6 ,, ...	6	,,
After 6 years	14	,,

In more than 53 per cent. of these alternating cases the deviation was seen before the end of the second year. This high proportion is due to the fact that the essentially alternating squints appear in early infancy.

Refractive error in cases of convergent squint.—In infancy and early childhood hypermetropia is the normal refractive condition, myopia or even emmetropia being then very uncommon. In the absence of the controlling influence of the fusion sense, the state of the refraction is the main factor in determining whether the eyes shall deviate inwards or outwards (see chapter iv.). It is not surprising, therefore, that convergent squinters are nearly always hypermetropic, and very frequently suffer from hypermetropic astigmatism also.

I have notes of 1,636 cases of convergent squint which are available for the present enquiry.

In twenty-three of these cases, or about $1\frac{1}{2}$ per cent., both eyes were myopic. I have not included these in the subjoined tables.

I have arranged the 1,384 cases of unilateral convergent squint in groups, according to the number of dioptres of hypermetropia in the lowest meridian of the fixing eye.

I have calculated the average refractive error in the highest and in the lowest meridian of each eye. In about three-fourths of these cases the age at which the squint was first seen was recorded on the case sheets. The table shows the number of cases found in each group, and the average onset-age.

The 229 cases of alternating convergent squint

TABLE I.

One thousand three hundred and eighty-four cases of unilateral convergent squint, arranged in groups according to the degree of refractive error in the lowest meridian of the fixing eye.

Group. Refractive error in lowest meridian of fixing eye.	Number of Cases.	Average age of onset.		Average refractive error.			
				Fixing eye.		Deviating eye.	
		Yrs.	Mos.	Lowest Meridian.	Highest Meridian.	Lowest Meridian.	Highest Meridian.
Not above + 1 D	83	2	10	0·45	1·36	0·48	1·7
Above + 1 D. Not above + 2 D	142	2	11	1·6	2·5	1·93	2·98
„ + 2 D. „ „ + 3 D	240	3	1	2·7	3·2	2·95	3·74
„ + 3 D. „ „ + 4 D	285	3		3·61	4·3	3·9	4·82
„ + 4 D. „ „ + 5 D	292	3		4·72	5·4	4·84	5·81
„ + 5 D. „ „ + 6 D	209	2	11	5·64	6·62	5·9	7·05
„ + 6 D. „ „ + 7 D	77	3		6·5	7·6	6·92	8·27
„ + 7 D. „ „ + 8 D	31	2	10	7·4	8·1	7·6	8·53
„ + 8 D. „ „ + 9 D	16	3	2	8·5	9·25	9	9·84
„ + 9 D. „ „ + 10 D	9	3	5	9·55	10·33	9·66	10·88

are similarly tabulated, except that they are arranged in groups according to the number of dioptres of hypermetropia in the right eye.

Astigmatism in unilateral convergent squint. —In the 1,384 cases I found the proportion of astigmatic to non-astigmatic eyes to be as follows :—

	Fixing eyes.	Deviating eyes.
No astigmatism ...	561	401
Astigmatism ...	823	983

But a very large proportion of people whose eyes are, for all practical purposes, normal, have at least half a dioptre of astigmatism. It would only be misleading to expect the eyes of squinters to conform to a higher standard than those of normal-sighted people. If we disregard astigmatism which does not exceed 0·5 D, we get the following results :—

	Fixing eyes.	Deviating eyes.
Astigmatism not over 0.5 D	836	628
Astigmatism over 0·5 D ...	547	756

On comparing the degree of refractive error in the two eyes in each of the 1,384 unilateral cases we find :—

Isometropia	427 cases.
Anisometropia	957 cases.

Or, disregarding differences which do not exceed 0·5 D—

Anisometropia not exceeding 0·5 D	663 cases.
Anisometropia exceeding 0·5 D	721 cases.

TABLE II.

Two hundred and twenty-nine cases of alternating convergent squint, arranged in groups according to the degree of refractive error in the lowest meridian of the right eye.

Group. Refractive error in lowest meridian of right eye.	Number of Cases.	Average age of onset.		Average refractive error.			
				Right eye.		Left eye.	
		Yrs.	Mos.	Lowest Meridian.	Highest Meridian.	Lowest Meridian.	Highest Meridian.
Not above + 1 D	33	0	11	0·6	0·81	0·63	0·79
Above + 1 D. Not above + 2 D	34	1	7	1·5	1·9	1·45	1·95
,, + 2 D. ,, ,, + 3 D	40	2	9	2·61	3·1	2·7	3·15
,, + 3 D. ,, ,, + 4 D	41	2	10	3·58	4·25	3·4	4·16
,, + 4 D. ,, ,, + 5 D	36	2	10	4·7	5·41	4·6	5·45
,, + 5 D. ,, ,, + 6 D	25	2	8	5·65	6·14	5·71	6·18
,, + 6 D. ,, ,, + 7 D	9	2	11	6·66	7·27	6·77	7·13
,, + 7 D. ,, ,, + 8 D	5	3	2	7·65	8·4	7·4	8·6
,, + 8 D. ,, ,, + 9 D	4	4	0	8·75	9·5	8·75	9·37
,, + 9 D. ,, ,, + 10 D	2	2	6	9·75	10·5	9·75	10·75

These *alternating cases* show a much lower percentage of astigmatic eyes than the unilateral cases :—

	Right eye.	Left eye.
No astigmatism	114	112
Astigmatism	115	117

If we disregard astigmatism which does not exceed 0·5 D, we find :—

	Right eye.	Left eye.
Astigmatism not over 0·5 D	140	141
Astigmatism exceeding 0·5 D ...	89	88

A comparison of the refraction of the two eyes in each of the 229 alternating cases shows :—

Isometropia	131 cases.
Anisometropia	98 cases.

Or, disregarding differences which do not exceed 0·5 D :—

Anisometropia not exceeding 0·5 D	185 cases.
Anisometropia exceeding 0·5 D	44 cases.

The relative frequency of squint was the subject of a careful enquiry which Mr. R. E. Hanson, oculist to the Education Department of the London County Council, has been kind enough to undertake at my request. During the last two years 10,239 school children have been examined in the Marylebone and the Tower Hamlets districts. 253 of these children exhibited a constant squint. In 231 of the cases the squint was convergent, and in 22 cases divergent. The test used was the mirror test described on page 80.

CHAPTER IV.

THE ÆTIOLOGY OF CONVERGENT SQUINT.

Many curious suggestions have, in former times, been made as to the cause of squint, such as "an evil disposition," naughtiness, imitating other members of the family who squint, the habit of turning the eye to look at a curl or ribbon on one side of the face, &c. The first definite theory, which was almost universally accepted, attributed convergent squint to a shortening of the internal recti. The natural corollary of this was that the affection might be cured by dividing these muscles or their tendons. The theory and its practical application seemed so plausible and simple that an age of indiscriminate muscle-cutting ensued. When the disastrous results of this practice were beginning to be recognised, Donders published his great work, and his "accommodation theory" of the cause of squint immediately came into vogue.

It will be well to examine these two theories in detail.

Muscle theory.—Many writers on convergent squint have attributed the anomaly to an undue

shortness or tightness of the internal recti, to a faulty insertion of the tendons of these muscles, or to a paresis of the external recti. To an observer who sees in a case of convergent squint only its most obvious symptom, viz., the abnormal convergence, it may seem reasonable to attribute this deformity to a defect of the muscles which move the eyes. A little investigation ought to convince anyone of the falsity of this view.

Of 1,523 cases of convergent squint in which I investigated this point, I found the power of abversion (outward rotation) of each eye tested separately to be quite perfect in 81 per cent.; in the remaining 19 per cent. the power of abversion was sub-normal, but the defect in most of these 19 per cent. was slight. This alone would seem sufficient to demonstrate that, in 81 per cent. of convergent squints at least, there is no undue shortness of the internal recti or paresis of the external recti muscles. In the 19 per cent. the deviation had, in the vast majority of cases, lasted several years, and the average degree of the defect of abversion was directly proportionate to the duration of the deviation. The defect of abversion in nearly all the 19 per cent. of cases would therefore appear to be due to *secondary* changes in the muscles and fasciæ, the result of the long continuance of the deviation, not its cause. One wonders that these secondary changes take place so seldom and to such a slight extent.

It is a matter of common observation that a convergent squint very frequently disappears, or the eyes even diverge, during general anæsthesia.

Occasionally a very high degree of convergence disappears when the accommodation is paralysed by atropine. It always reappears when the effect of the atropine has passed off.

Of cases of convergent squint which are treated with glasses alone, in about 30 per cent.[1] the eyes eventually become approximately "straight," and remain so as long as the glasses are worn.

These facts scarcely seem compatible with a muscular origin of squint.

Donders' theory.—When a person with normal emmetropic eyes looks at an infinitely distant object, such as a star, the visual axes are parallel, and the static refraction of each eye is sufficient to focus the image of the object on its retina. If the person now looks at an object only a foot away from him he must turn the eyes slightly inwards (convergence), in order that both visual

[1] I have no statistics of my own showing the proportion of "cures" effected by glasses alone, because I do not rely solely upon optical correction and operation. Of ninety-four cases of unilateral convergent squint quoted by Holthouse, the deviation was gradually overcome, and the eyes remained "straight" so long as glasses were worn in twenty-nine cases, or 30·8 per cent. Lang and Barrett (*R.L.O.H. Reports*, vol. xii.) had thirty-seven cures by glasses out of 102 cases, or 36·3 per cent. But in this series all cases in which less than five degrees deviation remained were considered cured.

axes may meet at the object. At the same time each eye must be focussed for near vision (accommodation), in order that the object may be seen distinctly. These two acts, accommodation and convergence, being always performed together, have become "associated" by hereditary habit, so that it is difficult to converge without accommodating or to accommodate without converging.

Hypermetropic eyes in a state of rest are out of focus even for distant objects, and still more so for near objects. A hypermetrope, therefore, in order to see distinctly, must accommodate in distant vision to a degree corresponding to the amount of his hypermetropia. In near vision he must accommodate both for the hypermetropia and for the nearness of the object. There is a *tendency* for a proportionate abnormal convergence to be associated with this abnormal effort of accommodation. Donders considered this tendency to be the cause of convergent squint, and he advised optical correction of the hypermetropia with a view to curing the squint.

By this chain of observation and reasoning the great physiologist let the first ray of light into this dark corner of ophthalmology, and gave the first indication for a rational treatment of convergent squint. But he was mistaken in supposing hypermetropia to be the fundamental cause of the malady.

The vast majority of children are hyper-

metropic. Of these hypermetropes only a small percentage present themselves at the clinics, whereas nearly all the squinters come at some time or other. Yet, even there, one sees at least a dozen hypermetropes who do not squint for one who does. This cannot be explained on the hypothesis that the severe cases of hypermetropia especially tend to cause squint, because statistics show that this is not the case (see tables i. and ii., chapter iii.).

It is usually stated that moderate degrees of hypermetropia are especially prone to be associated with squint. This is explained by saying that a child with moderate hypermetropia will accommodate in order to see distinctly. He only manages to make the extra accommodative effort by bringing into play also an associated effort of convergence. So that he sees distinctly with one eye and turns in the other. But, it is said, if the hypermetropia be excessive, he will not be able to accommodate sufficiently, so that he gives up the struggle, and neither sees distinctly nor squints.

But this ingenious argument is based on false premises. Moderate degrees of hypermetropia are more common than high degrees, not only in those who squint, but also in those who do not squint. I looked through my out-patient letters at the West Ham Hospital for about eighteen months, and tabulated all the cases

of hypermetropia, without squint, in which the refractive error was measured by retinoscopy under atropine. I tabulated the cases according to the degree of hypermetropia, and worked out the percentage number of eyes in each grade.[1]

The results, in the cases in which the hypermetropia was more than 2 D, were found to correspond very closely with my squint statistics treated in the same way. The proportion of cases with less than 2 D was lower among the non-squinters than among the squinters. Also the average degrees of astigmatism were higher among the non-squinters than among the squinters. This is evidently due to the fact that most of the non-squinters were patients who sought advice on account of headaches or visual defect.

It will thus be seen that the *degree* of the refractive error has very little to do with the question of whether the patient shall or shall not squint in the first instance, though, of course, when the squint is once established, the refractive error becomes a very important factor.

A high degree of hypermetropia has no in-

[1] In cases in which there was also astigmatism, the mean between the highest and lowest meridian was taken. I have since compared the results of taking the highest meridian only, also the lowest meridian only. The number of cases taken excludes any accidental source of error. A slight defect in this mode of comparison will, of course, suggest itself, but the error is not sufficiently grave to affect the main results.

fluence in determining an early onset of squint, as is conclusively shown by the following table. In the unilateral cases I have taken the average of the highest and lowest meridian in the fixing eye, and in the alternating cases I have taken the average of both eyes.

Less than +2 D	average	age	of onset	=	2·47	years.[1]
+2 D to +4 D	,,	,,	,,	=	2·85	,,
+4 D to +6 D	,,	,,	,,	=	2·92	,,
More than +6 D	,,	,,	,,	=	2·96	,,

A great many children who suffer from convergent squint have no more than the normal degree of hypermetropia, while 1 or 2 per cent. are actually myopic.

It is evident, then, that, though hypermetropia stands in some close ætiological relation to convergent squint, it is not the essential cause of the anomaly.

Ætiology of Convergent Squint.

For the sake of clearness, I will first state my proposition, and give the proofs afterwards.

In a case of convergent squint there is, in addition to the most obvious symptom, the deformity, always a defect of the fusion faculty, and there is nearly always a suppression of the vision of the deviating eye.

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[1] The average age of onset is low in these squinters, with less than 2 D of hypermetropia, because cases of essentially alternating squint are included in this table.

In the human infant, the motor coördinations of the eyes are already partially developed at birth. During the first few months of life these serve (in the absence of any disturbing influence) to maintain *approximately* the normal relative directions of the eyes. Soon the fusion faculty begins to develope. I have found distinct evidence of binocular vision in the sixth month. Normally the development of the fusion faculty is well-advanced by the twelfth month, and complete before the end of the sixth year. When the fusion faculty has begun to develope, the instinctive tendency to blend the images formed in the two eyes—the "desire for binocular vision," as it is called—will keep the eyes "straight." When the fusion faculty is fairly well developed, neither hypermetropia, nor anisometropia, nor heterophoria can cause squint. In fact, then, nothing but an actual muscular paralysis can cause an eye to deviate, in which case the resulting diplopia is intolerable. Sometimes, however, owing to a congenital defect, the fusion faculty developes later than it should, or it developes very imperfectly, or it may never develope at all. Then, in this case, there is nothing but the motor coördinations to preserve the normal relative directions of the eyes, and anything which disturbs the balance of these coördinations will cause a permanent squint. Thus the *essential cause of squint is a defect of the fusion faculty.*

In the presence of this fundamental cause, the eyes are in a state of unstable equilibrium, ready to squint either inwards or outwards on slight provocation. This provocation may be supplied by:—

(1) *Hypermetropia.* — As already explained, uncorrected hypermetropia causes a *tendency* to abnormal dynamic convergence of the visual axes. In the vast majority of cases of hypermetropia the fusion sense is perfect, so that this tendency is kept in check and the child does not squint. If, however, the fusion sense is deficient, the eyes are free to yield to this tendency, and a convergent squint is established. In the cases in which hypermetropia is the immediate exciting cause, the abnormal convergence is at first entirely dynamic, static convergence being *nil*. The squint is at first occasional—when the child is looking at nothing in particular, relaxing the accommodation, the deviation disappears. Optical correction of the refractive error at this period often cures the deviation. But if nothing is done, the excessive exercise of the function of dynamic convergence causes an abnormal static convergence to appear. So that the visual axes are convergent even when the eyes are completely at rest. At this period optical correction does not cause an immediate disappearance of the deviation. It may gradually do so, perhaps, after the glasses have been worn for a few weeks or months.

In cases in which the fusion sense is present but feeble, it may be strong enough to resist the strain of uncorrected hypermetropia during infancy, but may give way when the child's attention is directed to his first lessons. There is scarcely ever diplopia. Rarely one meets with a child who, when patiently questioned, admits that he sees a second image. This faint diplopia persists, but is never annoying.

During the first few weeks of life, even in cases of very high hypermetropia, the motor coördinations suffice to maintain approximately the normal relative directions of the eyes, until the developing fusion faculty takes control and makes everything safe. Probably the infant does not use his accommodation much at this early period. But, in rare instances, hypermetropia may cause a squint before the period at which the child should normally begin to acquire the fusion faculty (*e.g.*, Case A, 503, page 151). In such a case, if the deviation is allowed to persist, the natural development of the fusion faculty will, of course, be prevented.

(2) *Anisometropia*, and the rare congenital amblyopia, predispose to squint by making binocular vision more difficult. One occasionally sees a case of squint in which the fixing eye is approximately emmetropic and the deviating eye has perhaps 10 or 15 D of myopia.

(3) If the fusion sense be perfect, a want of

balance of the motor apparatus of the eyes will cause heterophoria but not squint. But in the absence of the controlling influence of the fusion sense, this motor imbalance is free to cause an actual deviation. This is seen when, in examining a case of heterophoria, fusion is temporarily rendered impossible by means of Maddox rod, coloured glasses, &c.

(4) *Specific fevers*, especially whooping cough, are often assigned by the parents as the cause of the squint. One usually hears that the child was seen to squint during convalescence, that the squint was, at first, occasional, and that it became constant after a few weeks. These children usually have a good deal of hypermetropia. The mode of origin of these squints is probably as follows:—The fusion sense is defective (shown by the absence of diplopia), but the motor coördination, or some slight degree of fusion sense, has hitherto sufficed to prevent the occurrence of a deviation. During convalescence the child is given picture-books. Owing to the muscular enfeeblement caused by his illness, he is unable to accommodate so well as formerly. The excessive effort of accommodation upsets the unstable equilibrium. At first the abnormal convergence is dynamic only, and disappears when the child relaxes his accommodation. A pair of spectacles at this period often brings about a cure. But if the opportunity be lost a static convergence

appears, and what was merely a premonitory occasional squint becomes constant.

As a contrast to these cases, it occasionally happens that a child, whose fusion faculty is perfect, suffers from a paresis of an external ocular muscle after diphtheria. The child complains of persistent and annoying diplopia until the muscle recovers its function. I recently had the opportunity of watching such a case. The patient, a little girl, aged five years, had paresis of the right external rectus muscle after diphtheria. She volunteered the statement that she saw "two nurses" and that "things looked funny." She was uncertain in her movements in running about, and often made a false shot in picking up a ball from the floor. When I gave her a picture book to look at, she covered the affected eye with her hand. When I saw her a few days later, she kept her head constantly turned to the right, evidently to enable her to blend the images. The case completely recovered in about seven weeks.

(6) *Violent mental disturbance*, caused by severe fright, "convulsion-fits," &c., may, in the absence of the fusion faculty, upset the equilibrium of the convergence centre. The convergence is static, it appears immediately, and is constant from the first. Refractive error is not an important feature. A large percentage of these cases are alternating.

Injury during birth.—Now and then one sees a case in which the power of abversion of an eye has been absent since earliest infancy, probably owing to injury to the sixth cranial nerve during birth. In most of these cases the

position of the eye prevents the natural development of the fusion sense. If no precautions are taken the eye may become extremely amblyopic.

Hereditary influence is a marked feature in any series of cases of convergent squint. In 1,373 cases of squint in which I was able to get probably reliable information, there was a history of squint in parent, grand-parent, brother, or sister of the patient in no less than 711, or 51·78 per cent.

Proof that the Essential Cause of Squint is a Defect of the Fusion Faculty.

If a pair of object-slides such as fig. 17 be put in the amblyoscope, a person with a normal pair of eyes will be able to blend the two imperfect images into one complete picture. If the angle of the instrument be varied the eyes will converge, or (to a certain extent) diverge also, in order to follow the objects and maintain fusion.

Now take a young patient with an ordinary unilateral convergent squint, and good vision in each eye. Adjust the amblyoscope to suit the angle of his deviation. He will only see with the fixing eye, the vision of the squinting eye being suppressed. If the suppression be now overcome by the method described in chapter viii., he will see the two imperfect images simultaneously. After a little practice, a position can usually be found in which the two imperfect images overlap, so that the patient sees them blended into one

complete picture. But they are blended only in that one position. If the angle of the instrument be again altered, the images at once separate—no effort can be made to maintain fusion. This shows that, though the fusion faculty is not quite absent, it is exceedingly ill-developed. The brilliant results obtained in these cases by training the fusion faculty at an early age, strongly support the view that the defect of this faculty was the fundamental factor in permitting the deviation to occur.

Consider next a typical case of essentially alternating squint. The visual acuity of each eye is perfect; there is no important refractive error; the movements of each eye separately are perfect. Why then does such a pair of eyes squint? The muscle theory cannot explain it, because there is no motor defect of either eye. Hypermetropia cannot be the cause, because there is little or no refractive error; moreover, the wearing of correcting glasses usually has no effect upon the deviation. No theory hitherto put forward has satisfactorily explained these cases. But, if the condition of the fusion faculty be examined, the mystery is cleared up at once. Let a patient with an essentially alternating squint look at a pair of object slides such as fig. 16 in the amblyoscope. When the objects are very far apart, he may be able to see both. But when they are made to approach each other, he loses sight of one of them.

No amount of practice will ever enable an essentially alternating squinter to see the two devices simultaneously when they are close together, much less to blend them. There is a congenital total absence of the fusion faculty.

The great frequency with which squint occurs in more than one member of a family has enabled me to obtain still more direct evidence. When I have had a child under treatment for convergent squint, I have, in very many instances, induced the mother to bring me also for examination a younger brother or sister of the patient, who had not hitherto squinted. I examined the fusion sense of all except the most intractable of these brothers and sisters of squinters with the amblyoscope. I have been able to follow the subsequent history of 157 of these children. In 106 cases I found the fusion faculty well developed. *Not one of these has subsequently squinted.* Of thirty-seven cases in which the note was "doubtful," six have since squinted. Of fourteen cases in which my note said "fusion faculty very deficient," eight have since developed a constant squint, and another child is said to cross her eyes occasionally,

CHAPTER V.

AMBLYOPIA, CONGENITAL AND ACQUIRED.

THE amblyopia discussed in this chapter is a partial blindness of an eye in which the most careful examination of the fundus and media reveals nothing sufficient to account for the defect. This amblyopia persists after accurate optical correction of any refractive error which may be present. It may be either congenital or acquired.

It will be well to consider congenital amblyopia and the acquired form separately, before discussing the amblyopia so often found in cases of convergent squint.

Congenital amblyopia, apart from squint, is very seldom met with. One should not accept a case as being one of congenital amblyopia, unless careful questioning of intelligent and observant parents makes it certain that the patient has never squinted as a child.

In the course of examining the refraction of many thousands of patients who have never squinted, I have only met with twenty-three cases of amblyopia, of $\frac{6}{18}$ or higher, which I have felt justified in regarding as congenital. These cases

are described in detail in the appendix (p. 219). They have certain peculiarities in common. The fundus and media are normal in appearance. The fields of vision, both for white and colours, are full. There is no scotoma. Central colour perception is normal. The peripheral form vision, up to within 20° of the fixation point, is normal.[1] So that the defect would seem to consist in a want of due preponderance of the macular region, and not in a general lowering of the sensibility of the visual apparatus. In no case was the vision of the amblyopic eye less than $\frac{6}{60}$. But the most remarkable feature of these cases is that the defect is confined to one eye, which almost invariably has a high degree of compound hypermetropic astigmatism; while the other eye has normal vision, and either normal refraction or hypermetropia, without any notable degree of astigmatism.[2] In many of the cases the fusion faculty was examined with the amblyoscope. It was found to be well developed.

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[1] For the purpose of testing the peripheral form vision, I use three metal screens about two inches square, each showing a white **O** on a black ground. The letters on the three screens are of different sizes. By a simple mechanical device the **O** is readily converted into a **C**. The screens are successively attached to the object-carrier of a perimeter. The investigation is proceeded with just as in mapping out the fields of vision, the patient being required each time to say whether the letter is **O** or **C**. In this way three zones are mapped out on the chart. This method though very valuable for purposes of scientific investigation, is too tedious for ordinary clinical use.

There is another small group of cases in which the amblyopia may almost certainly be regarded as congenital. Table iii., p. 76, includes only cases of squint in which my treatment was begun soon after the first appearance of the deviation and was carried out thoroughly, so that there was scarcely a possibility of any acquired amblyopia. It will be seen that the vast majority of these patients have, with optical correction, perfect vision in each eye. In seventeen cases out of the 193 the vision of the squinting eye was $\frac{6}{9}$ or $\frac{6}{12}$. In nine cases it was $\frac{6}{18}$ or $\frac{6}{24}$. In two cases it was $\frac{6}{36}$ or $\frac{6}{60}$. In no case was the vision lower than $\frac{6}{60}$, and in no case was the power of central

[2] I wish to avoid vague speculation. But the thought obviously occurs to one—Is it possible that, in some of these cases in which I have regarded the amblyopia as congenital, it may really have been acquired through the patient's confining his attention to the sharp image, even though this is fused with the blurred image of the astigmatic eye ? The fact that the amblyopia is always confined to one eye lends colour to the suggestion. The possibility of confining one's attention to the image formed in one eye, and yet, at the same time, exercising binocular vision, is easily demonstrated. Let one imitate the refractive condition of one of these patients by wearing a high cylindrical lens before one of one's own eyes, then read the test types with both eyes open. By confining the attention to the naked eye, one reads $\frac{6}{6}$. In reading a book one soon learns to disregard entirely the blurred image seen through the cylinder. At the same time, it is easy to convince oneself that one is exercising binocular vision—prism, base in, produces diplopia; prism, base out, causes convergence of the visual axes in order to blend images; four-dot test, &c.

fixation absent. On referring to my case books, I find that, in the more amblyopic cases, the defective eye nearly always had a high degree of compound hypermetropic astigmatism, while the other eye had perfect vision and nearly always simple hypermetropia without any notable astigmatism. These cases are described in the appendix.

Amblyopia acquired from disuse (amblyopia ex anopsia). In a case of convergent squint, even though the vision of each eye separately be perfect, the patient will, when both eyes are open, only see with the "straight" eye. The impressions received by the deviating eye are mentally "suppressed." In the case of a young child with a constant unilateral squint, the result of this disuse of the deviating eye is that its visual acuity gradually deteriorates. This deterioration from disuse is the more rapid the younger the child, so much so that it is commonly believed that an eye which squints in infancy is necessarily very blind. This is not so, as table iii. clearly shows. A child with good vision in each eye, who developes a constant unilateral squint at the age of six or eight months, will, in the absence of proper treatment, become rapidly blind in the squinting eye. This loss of vision in the infant's deviating eye is so rapid that the power of central fixation is often lost within eight or ten weeks. In an eye which begins to squint constantly at the age of, say,

eighteen months, the progress of the blindness is rapid, but much less so than in a younger child. At least five or six months usually elapse before the eye loses the power of central fixation. An eye which begins to deviate constantly at the age of three years seldom quite loses the power of central fixation in less than a year. I have never seen central fixation lost in a case in which the squint had first appeared after six years of age. After six years of age amblyopia ex anopsia seldom takes place to any great extent. Acquired amblyopia is a true loss of vision, not a failure of the function to develope, as is shown by cases quoted below.

I have notes of several cases in which ignorant or careless parents have accidentally performed upon their children most instructive (but disastrous) experiments bearing upon this subject. Here are brief notes of a few of the more striking examples.

Case A, 77.[1]—On November 14, 1895, I saw, at West Ham, a girl, aged 2 years 7 months. She had constant convergent squint R. E. 22°. The mother

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[1] The letters and numbers are the index marks of the notes in my squint case-books. In the case notes I have used certain abbreviations, some of which may require explanation, *e.g.*, "C.S.R.E. 22°" means "constant unilateral convergent squint, the angle of deviation being 22° in distant vision and the right being the deviating eye." "V.R.E." means "vision of the right eye." After glasses had been ordered, the angle of deviation and the visual acuity were always measured with the glasses on.

said that the child had squinted about a month. The squint was nearly alternating. After shading L. E. for a moment, she would fix with R. E., and continue to do so for a minute or two after removing the shade. Two hours after putting a drop of atropine into the L. E. she was seen to squint always with the L. E. and fix with the R. E.

November 21, 1895.—Retinoscopy under atropine each eye + 3·75 D sph. I ordered + 3 D sph. for constant wear; and a drop of liquor atropinæ to be put into the L. E. *only* every morning. Child to be seen again in a month.

August 22, 1901.—The child is brought to me at the West Ham Hospital. I have not seen her for nearly six years. The mother has no recollection of having used the drops for the L. E. She says the child wore the glasses for about a year, then lost them. No treatment since. Child is now aged 8 years 4 months. She has convergent squint R. E. 17°. The fixation of the R. E. is lost. Vision of the R. E. is reduced to the counting of fingers at 5 feet.

CASE D, 527. *October* 16, 1900.—Boy, aged 13 months, brought to Moorfields. The mother said he had "squinted the last few weeks." Child has convergent squint R. E. 30° about, varies slightly. Good central fixation R. E.

October 23, 1900.—Retinoscopy under atropine shows refractive error R. E. + 4 D sph. + 1 D cyl. ax. vert., L. E. + 4 D sph. Ordered glasses 0·5 D less than the retinoscopy. Ordered guttæ atropinæ 1 per cent. L. E. *only* every morning.

December 4, 1900.—Child uses unatropised R. E. always, both in near and distant vision, and squints with atropised L. E. Ordered, stop the drops and come again in a month.

January 8, 1901.—Child has now convergent squint. 18° with the glasses. The squint alternates. Ordered, wear glasses and come first week in April.

January 3, 1902.—Child has not been seen for a year. Soon after last visit mother says he lost his glasses. He has had no treatment since. There is now convergent squint R. E. 32°. R. E. has lost the power of central fixation. With L. E. bandaged, child sees the $1\frac{1}{2}$ inch ivory ball when it is rolling on the floor, but cannot find it when stationary, unless it is close to his feet.

CASE A, 432. *January* 11, 1900.—Boy, aged 2 years 2 months, was brought to me at West Ham Hospital. He had alternating convergent squint 27°. Squinted more in near vision. Mother said he had squinted less than a month. I ordered ung. atropinæ thrice daily, for both eyes, and told the mother to bring him again in a week to have his eyes tested for glasses.

November 7, 1901 (one year and ten months later). —The mother did not use the ointment and come again as directed, as the child's father " did not believe in having the eyes messed about with." Child now has convergent squint L. E. 34°. L. E. has lost the power of central fixation, and the ivory-ball test shows V. L. E. to be considerably less than $\frac{6}{60}$.

CASE D, 730. *July* 13, 1901.—Girl, aged 7 weeks, has convergent squint R. E. 10° approximately, angle varies slightly. Good central fixation R. E.

July 17.—Retinoscopy under atropine, each eye + 2·5 D sph. Ordered, ung. atropinæ for L. E. *only*, every morning.

July 31.—Child uses R. E. (unatropised) almost as often as L. E. (atropised). Ordered, continue atropine L. E. only.

August 28.—Atropine has not been used. R. E. now converges 30° about, fairly constant in degree. R. E. has lost the power of central fixation. Ordered, continuous occlusion L. E. for three days.

August 31.—No fixation R. E.

I did not care to order continuous occlusion of this

very young infant's better eye for many weeks, for fear of rendering it amblyopic. I therefore ordered the eye to be bandaged half of each day. In six weeks R. E. had regained steady central fixation.

CASE B, 24. *February* 4, 1896.—I was asked to see a girl aged 2 years 10 months. L. E. had squinted occasionally since she had whooping cough, aged $2\frac{1}{2}$ years, but for the last four or five weeks the squint had been constant. Angle of convergence 26°. After instilling atropine into R. E. only for about an hour, she turned in R. E. and used L. E. always, both in near and distant vision. She could easily see a small marble at the other end of the room with L. E. The sight of this L. E. must have been perfect or nearly so.

A week later, retinoscopy under atropine.—R. E. + 4·5 D, L. E. + 5·5 D.

I ordered spectacles for constant wear, R. E. + 4 D sph., L. E. + 5 D sph. I also ordered a drop of atropine to be put in R. E. *only* every morning, and proposed exercises with the amblyoscope in a few weeks.

The parents, not being favourably impressed by my methods, determined to have "another opinion." The child was taken to an ophthalmic surgeon who said she was too young for glasses. He ordered atropine for *both* eyes twice daily. This was kept up for rather more than a year, after which she was given spectacles. At the age of 7 years the surgeon operated on the left eye (tenotomy).

December 17, 1901.—The child was brought to me five years and ten months after I saw her first. She had convergence of L. E. 14°, while wearing her glasses. L. E. was prominent and caruncle sunk. The L. E. had lost the power of central fixation, and its vision was reduced to the counting of fingers at one foot from the face. I have since removed the deformity by advancement of the left external rectus, but the eye of course remains hopelessly blind.

CASE B, 83. *October* 27, 1897.—A girl, aged 2 years, was brought to me. The mother said she had squinted occasionally for several months, but she had squinted constantly since a few days before the August bank holiday. She had convergent squint L. E. 21°. Using R. E. she could always see the half-inch ivory ball at the far end of the (22 feet) room. When R. E. was bandaged she could see the $1\frac{1}{2}$ inch ball with the L. E., but she had great difficulty in finding the 1 inch ball unless she was allowed to begin to run after it before it had stopped rolling.

November 1, 1897.—Retinoscopy under atropine shows error of refraction to be, each eye + 3·5 D sph. + 0·75 D cyl. ax. vert. Ordered, spectacles 0·5 D less than the retinoscopy; continuous occlusion of R. E. for seven days; after that, one drop of liquor atropinæ to be put into R. E. *only* every morning.

December 7, 1897.—Child uses R. E. (atropised) in distant vision, and L. E. (unatropised) in near vision. Angle 17° with glasses. Ordered, continue.

February 8, 1898.—Child now uses (unatropised) L. E. always, both in near and distant vision, and squints always with (atropised) R. E. Ordered, stop using the atropine and come again in a month.

June 29, 1899 (one year and four months later).—Soon after the last visit the child's father, a bank manager, was transferred to a post in the north of England. The mother said that, as she was not able to bring the child to me, she thought she had better continue the drops. She used the drops, for the R. E. only, every day for about six months. Not since. The child has now constant squint R. E. 11°, with the glasses. Ivory-ball test shows vision of L. E. to be perfect, but vision of R. E. is barely $\frac{6}{60}$. Central fixation is present in R. E. but unsteady.

Of course all possible means have since been used to restore the sight of the R. E. The child has now learnt to read. On November 8, 1901, the vision of

the L. E. (which at first squinted) was $\frac{6}{6}$, and the vision of the R. E. (which was at first the fixing eye) was $\frac{6}{12}$.

CASE D, 332. On May 9, 1900, I saw, at Moorfields, a boy, aged 3 years 2 months. He had squinted constantly, R. E., since the age of 2 years 8 months. No heredity. Abversion perfect. C. S. R. E. 46°. Good central fixation R. E. Easily sees half-inch ivory ball at about 20 feet with R. E. Ordered, atropine both eyes for retinoscopy.

May 16, 1900. — C. S. R. E., with atropine, 37°. Retinoscopy R. E. + 7 D sph. + 1·25 D cyl. ax. vert. L. E. + 6·25 D sph. + 1·5 D cyl. ax. vert. Ordered, glasses 0·5 D less than retinoscopy; also, guttæ atropinæ 1 per cent. L. E. *only* every morning. To return in one month.

June 5, 1901 (thirteen months later).—Mother used drops for L. E. for a month, but was then ill, so child has since been neglected. The glasses have been worn constantly. C. S. R. E. 36°. R. E. has lost the power of central fixation. Ordered, continuous occlusion L. E. for one month.

July 3, 1901. — Central fixation, R. E., regained. Ordered guttæ atropinæ 1 per cent. L. E. *only* every morning for two months.

August 28, 1901.—Patient uses L. E. (atropised) in distant vision, and R. E. (unatropised) in near vision. Ordered, continue drops L. E. *only*, for two months.

December 4, 1901. — Drops have been used until three weeks ago, not since, as they were all finished. Child now, without atropine, uses the originally squinting R. E. always, and squints constantly with the originally fixing L. E.

April 2, 1902.—Last time I accidentally omitted to give the mother written directions; there was, therefore, some misunderstanding. Child now squints constantly with L. E. (the originally fixing eye) 24°. L. E

has central fixation, but very unsteady. Ordered, guttæ atropinæ 1 per cent. R. E. only, every morning for one month.

May 7, 1902.—Child will not use (unatropised) L. E. voluntarily even in near vision. Ordered, continuous occlusion R. E. for one month.

June 4, 1902. — Steady central fixation L. E. Ordered, discontinue pad and use guttæ atropinæ R. E. only, for two months.

August 6, 1902.—Child now uses L. E. (unatropised), and squints with R. E. (atropised), always in near vision, and usually in distant vision also. Ordered, stop drops.

August 30, 1902.—Child now uses either eye indifferently, alternating convergent squint 24°, with glasses.

CASE D, 286. *October* 9, 1900.—Girl, aged 5 years 7 months. She began to squint suddenly at the age of 3 years 9 months, and had squinted constantly ever since. Already under atropine. C. S. L. E., 36°. Retinoscopy, R. E. + 4·5 D sph. + 0·25 D cyl. ax. horiz.; L. E. same at approximate macula. Fixation lost L. E. Ordered glasses + 4 D sph. each eye. R. E. to be continuously occluded for one month.

August 9, 1901.—Case has been neglected, except that glasses have been worn. V. R. E. $\frac{6}{6}$, V. L. E. $\frac{4}{60}$. No fixation L. E. Ordered, continuous occlusion R. E. for three weeks.

August 30, 1901. — Treatment carried out. Good central fixation L. E. V. L. E. $\frac{6}{18}$. Ordered, guttæ atropinæ R. E. *only*, for six weeks.

October 4, 1901.—V. L. E. $\frac{6}{9}$. C. S. 16°. Child uses L. E. (unatropised) in near vision and R. E. (atropised) in distant vision. Ordered, continue atropine R. E. only, for two months.

December 3, 1901.—Vision $\frac{6}{6}$ each eye. Child uses L. E. and squints with R. E. always, both in near and

distant vision. Ordered, stop atropine and come in two weeks.

May 6, 1902. — Child has not been seen for five months. She has squinted constantly with R. E. (the originally fixing eye) during that time. V. R. E. $\frac{6}{18}$, V. L. E. $\frac{6}{6}$. Amblyopia to $\frac{6}{18}$ acquired in R. E. since last December. Ordered, guttæ atropinæ L. E. only, for one month.

June 6, 1902.—Squints now with L. E. (atropised) 18°. V. $\frac{6}{6}$ each eye.

NOTE. — This case is altogether exceptional, on account of the age (nearly seven years) at which amblyopia in the R. E. was acquired.

Amblyopia ex anopsia, like congenital amblyopia, concerns almost entirely the central and paracentral region of the retina, and produces no contraction of the peripheral limits of the field of vision. But the blindness often reaches an extreme degree which is never met with in the congenital form. In congenital amblyopia the central vision is never lower than $\frac{6}{60}$, the visual acuity normally found at 5° from the fixation point. In an extreme case of acquired blindness, on the other hand, there is often a scotoma extending about 25° to 30° round the centre of the field of vision. In this scotoma there may be bare perception of light. Outside this area fingers may be counted a foot or two from the face.

Amblyopia in Cases of Convergent Squint.

Congenital and acquired amblyopia having been studied separately, one is now in a position to

discuss the cause of the blindness so often found in cases of unilateral convergent squint. In any individual case, seen for the first time when the squint has lasted several years, it is impossible to say how much of the blindness may be due to disuse of the deviating eye, and how much may be congenital. Statistics, however, enable one to draw a very accurate general conclusion.

Tables iii., iv., and v., show the visual acuity of the deviating eye in cases of constant unilateral convergent squint. I used Snellen's types or the ivory-ball test at the first visit, when possible, and on many subsequent occasions. Nearly all these children have since become old enough to allow me to confirm the results of the ivory-ball test by Snellen's types. The visual acuity noted in the tables is the final result, with optical correction, and after all possible means had been used to remove any acquired amblyopia.

I have included in the tables only cases in which I could be reasonably certain as to the time of onset of the deviation, and in which my directions were subsequently followed to my satisfaction. The cases had either received no treatment before I first saw them, or they had merely been given glasses. Some had been operated upon.

Any defect of vision found in the cases in table iii. may be considered as congenital. In

these recent cases, any amblyopia which might have been acquired would certainly have been removed by the subsequent treatment.

Table iv. shows the visual acuity of the deviating eye after all possible means had been used to remove any defect which might be present. The vision was at first, in many cases, very considerably lower than shown in the tables.

In the cases in table v. the squint had, in most cases, lasted so long that no improvement in vision was possible.

A comparison of table iii. with table v. shows that congenital amblyopia only occurs in a very small proportion of the cases, and is never responsible for the extreme blindness so often found in neglected cases of unilateral squint.

TABLES SHOWING THE VISUAL ACUITY OF THE DEVIATING EYE IN 787 CASES OF CONSTANT UNILATERAL CONVERGENT SQUINT.

TABLE III.

Cases which I saw first when the patient had squinted during less than one-eighth of his or her life.

Vision of the deviating eye.	Age of onset of the deviation.			Total.
	Before 12 months.	1 to 3 yrs.	After 3 yrs.	
$\frac{6}{6}$	23	62	80	165
$\frac{6}{9}$ and $\frac{6}{12}$	2	6	9	17
$\frac{6}{18}$ and $\frac{6}{24}$	1	3	5	9
$\frac{6}{36}$ and $\frac{6}{60}$	0	1	1	2
Less than $\frac{6}{60}$	0	0	0	0
Fixation lost irrecoverably	0	0	0	0

TABLE IV.

Cases which I saw first when the patient had squinted during more than one-eighth and less than one-half of his or her life.

Vision of the deviating eye.	Age of onset of the deviation.			Total.
	Before 12 months.	1 to 3 yrs.	After 3 yrs.	
$\frac{6}{6}$	5	17	51	73
$\frac{6}{9}$ and $\frac{6}{12}$	3	26	32	61
$\frac{6}{18}$ and $\frac{6}{24}$	0	14	14	28
$\frac{6}{36}$ and $\frac{6}{60}$	0	5	9	14
Less than $\frac{6}{60}$	0	1	4	5
Fixation lost irrecoverably	0	2	5	7

TABLE V.

Cases which I saw first when the patient had squinted during more than one-half of his or her life.

Vision of the deviating eye.	Age of onset of the deviation.			Total.
	Before 12 months.	1 to 3 yrs.	After 3 yrs.	
$\frac{6}{6}$	0	3	11	14
$\frac{6}{9}$ and $\frac{6}{12}$	2	7	19	28
$\frac{6}{18}$ and $\frac{6}{24}$	4	32	54	90
$\frac{6}{36}$ and $\frac{6}{60}$	8	53	41	102
Less than $\frac{6}{60}$	55	103	21	179
Fixation lost irrecoverably	56	110	25	191

When I have been consulted about a case of squint, I have always warned the parents of the patient that, in the event of a younger member of the family developing a squint, the case ought to receive attention without delay. I have, therefore, been fortunate enough to see an unusually large proportion of my cases soon after the first appearance of the deviation.

It is remarkable that only eight cases, commencing before twelve months of age, appear in table iv. This is evidently because parents who have been warned, and those who are especially solicitous for the welfare of their children, seek advice immediately. These cases are included in table iii. The other infants are usually left without treatment for several months, so that they appear in table v.

CHAPTER VI.

THE METHOD OF INVESTIGATING A CASE OF SQUINT.

Every case of squint should be systematically investigated, as it is only by a thorough knowledge of each case that a rational line of treatment can be determined upon. The following is the plan I always use. It may appear rather formidable at first sight, but, with practice, one can carry out the various tests with great rapidity and precision. The time will surely not be grudged when it is remembered that, in cases which are presented early enough, the patient's whole future career may depend upon the skill and care of the surgeon who first sees the case.

(1) History.

(2) The character of the squint

(3) The power of fixation in the deviating eye.

(4) Movements of each eye separately. Dynamic convergence.

(5) Vision testing.

(6) The condition of the fusion faculty.

(7) The angle of the deviation.

After using atropine for from three to eight days :—

(8) The refraction.

(1) History.—Under this head should be noted : (*a*) Age of onset, when it can be determined. It may often be fixed very precisely by reference to some family event, such as the birth of the next child. (*b*) Mode of onset. Whether it began as an occasional squint or was constant from the first. (*c*) Any illness or injury immediately preceding the appearance of the deviation *e.g.*, whooping cough, measles, a blow on the head, "fits," severe fright, &c. (*d*) Evidence of heredity—squint in brother, sister, or parents of the patient.

(2) The Character of the Squint.—The presence of a deviation and its character, whether convergent or divergent, unilateral or alternating, may often be determined by simple inspection. But appearances are sometimes misleading, *e.g.*, the high angle gamma often found in hypermetropes may simulate a divergent squint or mask a slight convergent squint. The low, or even negative, angle gamma usually associated with myopia may give an appearance of abnormal convergence or mask a slight divergence.

The cover test is at best only a rough test having many sources of error. It is, moreover, not possible to use it with young children. But, as it is very generally employed, it will be well to describe the

proper method of making the test, and the fallacies to be guarded against.

Tell the patient to look steadily at some distant object. Take a narrow card or folded paper and, with a rapid lateral movement, cover, say, the patient's left eye, taking care not to touch his face. If the right eye makes no movement it was probably[1] not squinting. Now uncover the left eye, and see that the patient looks steadily at the distant object. Screen the patient's right eye in the same way. If the left eye makes no movement it was probably[1] not squinting either. If, however, when one eye is covered, the other has to make an outward movement in order to fix the object, it was previously squinting inwards; if it rotates inwards it was squinting outwards.

In a case in which the patient has been shown to have a squint, but in which he can fix with either eye at pleasure without the other eye being screened, and can maintain fixation after a momentary closure of the lids, the squint is alternating.

In some occasional squints and heterophorias, while both eyes fix truly when uncovered, either eye deviates when screened, but immediately recovers itself on removal of the screen.

The mirror test.—This is an entirely satisfactory test, and can be used quite well even in the case of the youngest infants. The patient should be in the dark room, with the lamp behind him. The light is reflected from the mirror of an ophthalmoscope, from a distance of about 2 ft., into the patient's eyes. An infant will immediately look at the mirror; an older patient should be

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[1] If the eye has lost the power of central fixation, it may perhaps make no movement, or it may move in a direction which would mislead any but a careful observer.

told to do so. A tiny image of the ophthalmoscope mirror is formed on the patient's cornea. Owing to the angle gamma, this reflection of the mirror is usually slightly to the nasal side of the centre of the pupil. By flashing the light rapidly from one eye to the other, any want of symmetry in the position of the reflections is at once detected. It may easily be seen, too, which is the deviating eye, and, with practice, a very good guess as to the extent of the deviation may be made.[1]

Squint or paralysis.—In the case of a patient who is old enough to speak, the persistent diplopia would prevent one mistaking a case of paralysis of one or more of the external ocular muscles for a case of comitant squint.

But, in the case of an infant who has recently suffered from diphtheria, an objective test is required. The patient is on the nurse's knee in the dark room, with the light behind him. The nurse holds his head immovable. The light is thrown into his eyes, from an ophthalmoscope mirror, from a position slightly to one side of him. When he fixes the mirror the approximate

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[1] According to Hirschberg, when the reflection on the squinting eye is at the margin of the cornea the angle of the deviation is about 45°, when it is at the margin of an average-sized pupil the angle is about 15°. Allowance should be made for the estimated size of the angle gamma.

degree of his deviation is noted. The light is next thrown into his eyes from the other side. If, when he again fixes the mirror, the angle of the deviation is greater or less, we have to deal with a paralysis or paresis, and not with a comitant squint.

In carrying out the test, it is essential that the light be thrown into the eyes in both cases from approximately the same level, as, in many cases of true squint, the eyes converge more on looking down and diverge more on looking up.

(3) The Power of Central Fixation.—An exceedingly important point, in its bearing on the treatment and prognosis of a case of unilateral squint, is the presence or absence of the power of central fixation in the deviating eye.

The patient being in the dark room, with the light behind him, throw the light from an ophthalmoscope mirror first into his good eye, while he looks at the mirror. Note the position of the reflection of the mirror on the cornea of this eye. Then cover the good eye, and note whether the previously deviating eye can now fix the mirror so as to bring the corneal reflection into a corresponding position. If it does so, there is central fixation. There may be no fixation, in which case the eye wanders. Or there may be false fixation, in which case an eccentric part of the retina is used for the purpose. An eye which at first appears to have lost fixation may some-

times, with a little patience, be induced to fix correctly.

In the clinics one frequently sees some such procedure as the following: The surgeon covers the patient's good eye, then asks him to follow the movement of his finger with the deviating eye. If he is able to do so, it is assumed that he has central fixation in this eye. This is entirely fallacious. If the deviating eye has any sight at all it can usually follow the movements of a large object. If the deviating eye has false fixation in a position of only slight convergence, even the most prolonged examination by this method will certainly lead to error.

(4) THE MOVEMENTS OF EACH EYE SEPARATELY should be tested by covering one eye and getting the patient to look with the uncovered eye from side to side and up and down. The test is then repeated with the other eye. This may usually be done even in the case of young infants by showing them something in which they are likely to take an interest. If each eye can be separately abverted until the edge of the cornea touches the outer canthus, abversion may be considered full. The power of adversion varies considerably within normal limits. Most people can advert each eye separately until the corneal margin is within less than one-tenth of an inch of the caruncle.

DYNAMIC CONVERGENCE.—A careful distinction should be made between static and dynamic convergence, just as static and dynamic refraction are distinguished from each other.

When a person looks at a distant object, if he has no squint the static convergence of the eyes is *nil.* If he has a convergent squint there is a static convergence corresponding to the angle of the squint. If he has a divergent squint, the static convergence is a negative quantity.

If now he fixes a near object, there is superadded to the static convergence a dynamic convergence.

A person who in distant vision has no squint, will usually exercise just sufficient dynamic convergence in near vision (in association with dynamic refraction, or accommodation) to allow him to fix the object correctly with both eyes. If, however, his dynamic convergence be excessive, the eyes will tend to squint inwards in near vision. A perfect fusion-sense will keep this tendency in check. But if the fusion-sense be defective, this tendency will be free to cause a squint in near vision. In a case of divergent squint, when the fixing eye accommodates for a near object the divergent eye will usually recover itself to a certain extent. In convergent squint, the faulty eye should turn in still more towards the nose in near vision. As will be seen later, the estimation of the dynamic convergence is of the utmost importance in deciding the question of operation for squint.

Method of estimating the dynamic convergence in a case of squint.—Stand at arm's length from the patient. Shut one eye and hold the

point of the finger, or perhaps some more attractive object, in line with the open eye and the patient's fixing eye. Let the patient look at the object while it is gradually brought nearer his fixing eye. The fixing eye will remain immobile, the deviating eye manifesting the convergence of both. In this way, the slightest movement of convergence of the deviating eye can at once be seen, and the point at which the deviating eye begins to diverge again can be accurately noted. This simple procedure should never be omitted.

(5) VISION TESTING.—(*a*) The vision of patients who are old enough to read letters should be tested with *Snellen's distant types*. This would appear a very simple matter, yet it is surprising how often, in the clinics, one finds the vision of the better eye recorded as the vision of each eye, even by experienced assistants. The desire to use the accustomed eye is so strong that, when this is shaded, the patient will involuntarily screw his head round to try to see past the shade.

(*b*) *Ivory-ball test for young children.*—Apart from its scientific interest, it is often of great practical importance to be able to estimate approximately a young child's visual acuity. For this purpose I use five little ivory balls varying in size from half an inch to one and a half inches in diameter. The child is first allowed to handle the balls with both eyes open. Then one eye is covered

by a pad, or, if, he wears glasses, by a piece of cotton wool stuffed behind the lens. He is then asked to go and pick up the balls as they are thrown on the floor to a distance of six or seven yards, one by one, beginning with the largest. By spinning the ball in the fingers as it is thrown, it can be made to "break" on touching the floor, so that it does not go quite in the direction in which it appeared to have been thrown. It is easy to tell, by the way in which the child runs for the ball, whether he really sees it before he starts or is only going to look for it. I test the presumably better eye first, so as to give the other eye the benefit of experience.

Children are always ready to play this ball game. This method of vision-testing only takes a few minutes, and it succeeds with most children who are old enough to walk. I have used it since 1896, and, in cases in which I have subsequently been able to test the vision by means of Snellen's types, I have found my conclusions confirmed.

(6) The Condition of the Fusion Faculty may, as a matter of convenience, be investigated at this point, but it is usually better to defer the examination until the state of the refraction has been ascertained and the effect of the mydriatic has passed off.

Examination with the *amblyoscope* I have found by far the most rapid, precise, and reliable method of ascertaining the condition of the fusion sense.

Of late years I have used it to the exclusion of all other methods. It is fully described in chapter viii.

I wish again to draw attention to the distinction between the possession of the fusion faculty and the presence of binocular vision. A patient may have a convergent squint of very high degree, with suppression of the vision of the deviating eye, and yet training with the amblyoscope may have perfectly developed his fusion faculty. In this case, if the patient looks into the amblyoscope while the instrument is adapted to the angle of the squint, he fuses the pictures —his fusion faculty finds its expression in the act of binocular vision. The same thing happens when his eyes are put approximately "straight" by operative or other means.

Javal, Maddox, and others estimate the "depth of suppression" by the ease or difficulty with which diplopia may be artificially elicited. Diplopia may sometimes be elicited by placing a red glass before the better eye and alternately covering and uncovering it while the patient looks at a candle flame. There is a fallacy here which must be guarded against—the patient recognises the fact that he sees a red light with the "good" eye, and that, when this is covered, he sees a white light. He therefore often says he sees two lights, a red and a white, even though he does not see them both simultaneously. If the red glass fails to produce diplopia, success may often be attained by placing a *horizontal* prism before the deviating eye, so as to throw the image of the flame on a part of the retina nearer the macula.

Some writers have recommended, as a test, placing a prism *vertically* before one eye, to throw the false image out of what they call the "band of suppression." Diplopia can almost always be elicited in this way, even in cases of total absence of the fusion sense. The explanation of this is not far to seek. The conjugation of the two eyes in horizontal movements, being intended to subserve the act of binocular vision, was probably acquired at no very early period in the development of the human race. The conjugation of the eyes in vertical movements, on the other hand, I believe we share to a great extent with most of the mammals. It is not surprising that disturbance of this very ancient function, by vertical displacement of the image in one eye, should produce diplopia.

But diplopia tests really give very little information as to the condition of the fusion sense. Some cases, in which diplopia is with difficulty elicited by these methods, readily get the highest grade of binocular vision after fusion training with the amblyoscope, and are subsequently completely cured. Others, again, who easily see double, may be incapable of acquiring more than grade 1 binocular vision.

7) The Measurement of the Angle of the Deviation.—At the first, and at each subsequent visit, the angle of the deviation is accurately measured. When the child has been ordered glasses, the measurement is always made with the glasses on. The case-sheet then shows at a glance the progressive effect of the treatment upon the deviation, and helps one to decide whether it is advisable to supplement this treatment by operation.

Four methods of measuring the angle of deviation are here described.

(*a*) *The deviometer*.[1]—This instrument can be very rapidly used, no adjustment being required. Measurements obtained by it are very accurate. It can be used quite easily even with the youngest children.

A wooden stand, about 10 inches high, supports a horizontal wooden arm, 2 inches wide, $\frac{1}{4}$ inch thick, and about 2 feet long. This arm is pivoted at the end, so that it may be swung over to either side as required. The arm is painted black in front. On the back is a scale of tangents to degrees at 60 centimetres distance. A flat, hook-shaped piece of brass, having a white spot on it, slides along the arm. In front of the pillar, below the zero of the scale, is a specially made electric lamp, 5 inches high and $\frac{3}{4}$ inch in diameter. Flexible wires go from the electric lamp to the wall plug. An electric bell-push is used instead of a switch, so that, by pressing the button, the light may be flashed on and off very rapidly. A string 60 centimetres long is attached to the upright pillar of the instrument. At the end of the string is a ring.

Measurements and details of construction of the deviometer will be found in the appendix.

The instrument is put on a table. The nurse

[1] I have no wish to claim originality for this instrument. The principle is that of Maddox' scale and Priestley Smith's tape, the former of which I used for years before I devised the deviometer.

sits at the table with the child on her knees. She puts the ring on her finger, and holds the child's head steady with her hands, keeping the string taut. The surgeon "sights" the child's eyes through the nick in the top of the stand, and

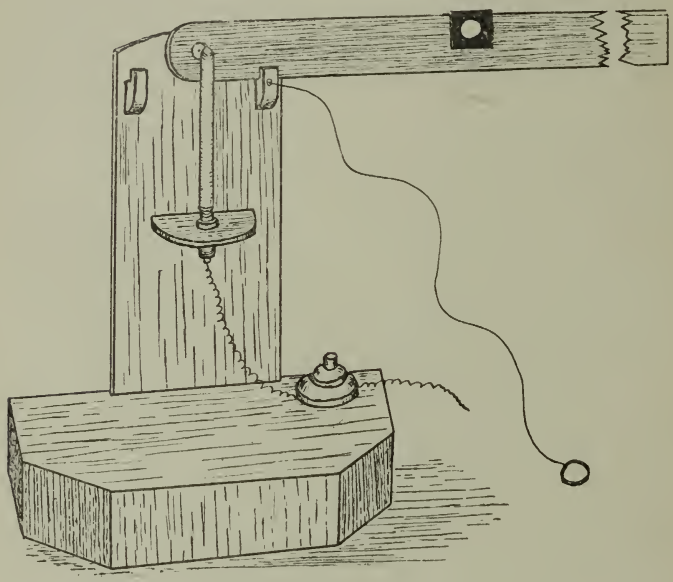

FIG. 9.

presses the button. The child immediately looks at the light with his fixing eye. The reflection of the lamp forms a vertical line of light on the cornea of this eye, which shows the correct position of fixation. The position of the line of light on the cornea of the squinting eye enables a good guess to be made as to the angle of the deviation. The light is discontinued. The brass

traveller with the white disc is slid along the arm to the position which corresponds to the guess. The brass traveller being tapped with the finger, the metallic sound causes the child to look at it. If it does not, a lighted match, held in front of the traveller, will always attract his attention. The button is then pressed, and the light flashed on for an instant. If the line of light on the cornea of the squinting eye is in a corresponding position to that which it formerly occupied in the fixing eye, the angle of the squint is read off on the scale on the back of the arm. If not, the traveller is moved a little, and when the child looks at it the light is flashed on again, and so on till the true position is found.

An older patient can, of course, sit at the table and hold the string himself, and look at the zero of the scale, or the white disc on the traveller, when directed to do so.

(*b*) *Maddox' tangent scale* (fig. 20, page 171) is an admirable device for measuring the deviation. The method is very rapid and accurate. It is not, however, easy to use with small children. I always use it at Moorfields, because the apparatus is fixed in the wall out of harm's way, and there is nothing to get out of order, however roughly it may be used.

The large figures are used for measuring heterophorias with the Maddox rod. With these we are not at present concerned. The small

figures, in the horizontal scale, represent tangents to degrees at a distance of one metre. They are printed on a strip of paper, which is pasted on a board about seven feet long. In the centre of this scale is a candle. Below the candle is attached a light bamboo rod one metre long.

The patient rests his cheek against the end of the metre rod. The surgeon puts his head below the rod, so that his eye is vertically below the rays of light which pass from the candle to the patient's face. The patient is first told to look at the light. The position of the image of the candle flame on the cornea of the fixing eye is noted. A guess is made as to the angle of the squint. The patient now is told to look at the figure which represents the guess. If this is too much or too little, other figures are named till the reflection of the candle flame on the cornea of the deviating eye occupies a position similar to that which it formerly occupied in the fixing eye.

(*c*) *Priestley Smith's tape method.*—The advantages of this method are that it takes very little time and the apparatus required is simple. It is moderately accurate. It is, however, not easy to use with young children.

A string one metre long has a ring at one end. To the ring is attached a graduated tape. The tape has a weight at its other end. The patient holds the free end of the string against his temple.

The surgeon puts the ring on a finger of one of his hands in which he holds an ophthalmoscope mirror. The tape is allowed to slide between the fingers of the other hand, the weight keeping the tape taut. The patient is first told to fix the mirror, while the light of a lamp is reflected into the fixing eye. The position of the image of the mirror, on the cornea of the fixing eye, is noted. The light from the mirror is now thrown on to the deviating eye, and the patient is directed to look at the surgeon's tape hand. This is moved horizontally, till the position of the image of the mirror, on the cornea of the squinting eye, is similar to that which it formerly occupied on the cornea of the fixing eye. The string keeps the ophthalmoscope hand at one metre from the patient's eye. The observer keeps the tape hand as nearly as possible at the same distance from the patient's eye. The graduated scale on the tape, where it slides through the tape hand, shows approximately the angle of the deviation in degrees.

If the length of the string be made 60 cm. instead of one metre, an ordinary Continental tape measure may be used for the graduated tape, one centimetre representing approximately one degree.

(*d*) *The perimeter method.*—I describe this because a perimeter is to be found in every eye clinic and ophthalmic surgeon's consulting room. The patient is seated at the perimeter, which is adjusted so as to bring his deviating eye *accurately*

in centre of the arc. A candle is placed at the far end of the room, in line with the zero of the perimeter and the patient's deviating eye. He is told to look steadily at this candle with his fixing eye. A second candle or taper, with the eye of the surgeon looking exactly over the top of the flame, is carried round the arc of the perimeter till the reflection of the flame lies in the centre of the cornea of the deviating eye. The position of the taper on the graduated arc of the perimeter, shows the angle of the squint in degrees.

This is a wretched method. It is inaccurate, as it takes no account of the angle gamma. It cannot be used in the case of a small deviation, as the surgeon's head then prevents the fixing eye seeing the distant candle. It cannot be used for young children. The preliminary arrangement takes up so much time that a surgeon who relies upon this method is apt to neglect to measure the squint at all.

The angle gamma may be measured separately, if the patient's squinting eye has not lost the power of central fixation. Cover the fixing eye. Let the patient steadily fix the zero of the perimeter with the squinting eye. The taper with the surgeon's eye looking exactly over it, is carried along the arc till the reflection of the flame appears in the centre of the cornea. The position of the taper on the graduated arc shows the size of the angle gamma. This angle gamma should be added to the perimeter measurement of a convergent squint and subtracted from that of a divergent squint.

(8) The Refraction.—This is examined by retinoscopy, after atropine has been used, for both eyes, three times a day, for from three to eight days. For young children I prefer the atropine ointment, 1 per cent. Atropine drops, if used too freely, occasionally cause unpleasant symptoms, whereas the ointment almost never causes trouble. The nurse or mother should be shown how to insert the ointment within the lower eyelid with a glass rod. One now and then sees a young child in whom atropine produces perfect cycloplegia with only very slight mydriasis.

Children of two years and upwards may, with a little tact, usually be induced to allow one to put on a trial frame and proceed with retinoscopy in the usual way.

In the case of a very young child, a different plan must be followed. The nurse sits in the dark room with the child in her arms. The light is placed above and behind the child's head. The light being reflected from the ophthalmoscope mirror into the child's fixing eye, he immediately looks at the mirror. The lenses are handed, one by one, to the nurse, who holds them up before the fixing eye, or the surgeon may hold the lenses himself. There is seldom much difficulty so far. Now, in order to investigate the deviating eye, it is necessary to screen the fixing eye. The child may, perhaps, not tolerate any screen held near his face. In this case, I hold a black cardboard

screen, about halfway between the child's face and my own, in such a way as to cut off the view of the mirror from the child's fixing eye. The light now being thrown into the deviating eye, the child looks at the mirror with this eye. If central fixation in the deviating eye of one of these very young children has already been lost, one can only make an approximate guess as to the refractive error of this eye. A glass may then be ordered provisionally, to be exchanged for an accurate correction when one has succeeded in restoring central fixation.

In the case of an older patient, one can measure very accurately the refractive error of an eye which has lost fixation. Throw the light into the fixing eye, and note the position of the reflection of the mirror on the cornea, while the patient fixes the mirror. Now direct the patient to look at a small white card, which is held three or four feet away from him by an assistant. Throw the light on to the cornea of the deviating eye, and manœuvre the card till the reflection of the image on the cornea is in a position corresponding to that which it formerly occupied in the fixing eye. Now proceed with the retinoscopy.

CHAPTER VII.

THE TREATMENT OF CONVERGENT SQUINT.

I PROPOSE to describe first the treatment I adopt in cases of constant unilateral squint. Any modification of treatment required in occasional or alternating cases will be considered afterwards.

The objects to be kept constantly in view in the treatment of squint are—(*a*) To prevent deterioration of the vision of the deviating eye, and to restore, as far as possible, the sight of this eye in cases in which amblyopia from disuse has already been allowed to occur. (*b*) To endeavour to remove the fundamental cause of the squint, by training the fusion sense at the earliest possible age. (*c*) To restore the visual axes to their normal relative directions.

There are five therapeutic measures at our disposal, any or all of which it may be necessary to use in our endeavour to attain these objects. (1) Optical correction of any refractive error which may be present. (2) Occlusion of the fixing eye. (3) Instillation of atropine into the *fixing* eye *only*. (4) Training the fusion sense. (5) Operation.

(1) Optical Correction.—As has been demonstrated in chapter iv., the essential factor which allows a deviation to occur is a defect of the fusion faculty. The eyes then, being uncontrolled by the necessity for fusion, are for a time kept approximately "straight" by their motor coördinations. But they are in a state of unstable equilibrium, and are ready to squint, either inwards or outwards, in response to influences which would have no effect if the fusion faculty were normal. In a very large proportion of the cases it is the state of the refraction which chiefly determines whether the eyes shall deviate inwards or outwards. Thus, in the great majority of cases, the eyes of hypermetropic squinters deviate inwards, and the eyes of myopic squinters deviate outwards. It is rational treatment, therefore, to attempt to overcome the deviation by optical correction of any refractive error which may be present.

In cases of simple hypermetropia, or hypermetropic astigmatism, or compound hypermetropic astigmatism, my usual practice is to order spectacles fully correcting any astigmatism which may be present, and correcting all but 0·5 D of the hypermetropia. The reason for the slight under-correction of the hypermetropia is this:—When the effect of the atropine, used for the retinoscopy, has passed off, some of the hypermetropia will in any case become "latent," so that

fully-correcting glasses, which gave perfect distant vision under atropine, will, when its effect has passed off, make all distant objects appear misty. This blurring of distant objects not only shortens the child's range of vision at a time when the acuity of the physical senses has the most marked effect upon his mental development, but the effort to see distinctly actually appears to affect unfavourably the angle of the deviation.[1]

In a case of mixed astigmatism, the refractive error should be exactly corrected.

A certain percentage of cases of convergent squint are myopic. Any myopic astigmatism should of course be exactly corrected. But as regards simple myopia, it might naturally be thought that a considerable under-correction would tend to lessen the abnormal convergence by preventing any effort of accommodation even in near vision. I began by acting upon this assumption, but was gradually forced by experience to abandon it. I find that the best results are obtained by exactly correcting any myopia and myopic astigmatism which may be present. Myopes who begin early to wear fully-correcting

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[1] I have taken over a considerable number of hospital squint cases which were formerly under the care of a colleague, who used habitually to over-correct the hypermetropia to the extent of 1 D. In the majority of these cases the deviation became less within a few weeks of ordering glasses with which the patients could see distinctly.

glasses, use them quite comfortably for all purposes, and appear to have as good a range of accommodation as emmetropes.

In a case of anisometropia, the refractive error of each eye should be corrected according to the preceding rules. This applies even to cases in which one eye is hypermetropic and the other myopic.

When glasses are ordered, it is a good plan, especially with young children, to continue the atropine, which has been used for the retinoscopy, until the glasses arrive from the optician. It should then be discontinued. Even an infant soon discovers that he sees better with the glasses than without them, and by the time the effect of the atropine has passed off, the wearing of glasses has become as much a habit as the wearing of clothes.

The glasses should be worn constantly, except when the child is in bed at night. They should never be removed at any other time, except for toilet purposes.

In the case of children who are old enough to attend school, some surgeons order one pair of glasses for distant vision and a stronger pair for reading. I have tried this plan and found it most unsatisfactory. In changing from one pair to the other there is often a considerable interval in which no glasses are worn at all. Besides, the exercise of a *normal* amount of accommodation in

association with dynamic convergence, in near vision, is a physiological act, and its suspension has not appeared to me to have permanently lessened the angle of the squint.

The quality and fit of the spectacle-frames are very important. Steel is the best material for children. Steel frames of good quality will remain without rust for a long time, if the child be kept moderately clean. After a time, the growth of the child's face will necessitate larger frames. For infants and young children the lenses should be circular,[1] or oval with their long axes vertical, in order that there shall be no temptation for the child to look over them. The lenses should be as large as possible, and should be centred for distant vision. They should be as close to the eyes as they can be without touching the lashes. The bridge piece, which arches over the bridge of the nose, should be of tempered steel, broad, flat, and strong, and should be very accurately fitted. A broad thin plate of tortoise-shell, carefully fitted under the arch of the bridge piece, prevents rust and distributes the pressure over a larger area. Spectacles for children of

[1] Circular lenses, however, have this disadvantage—unless the spectacles are carefully made, the lenses are apt to rotate in the frames, so that the axes of cylinders become displaced. This may be prevented by putting a little Canada balsam in the groove of the rim. If it should be necessary, at any time, to replace a cracked lens, the balsam may be melted by a moderate heat.

3 years and upwards should have flexible curl sides to hook behind the ears.

Infants and very young children should have their glasses tied on. The sides, in this case, should be straight and should have a loop at the end. They should be very short, only reaching to just above the ear. About $\frac{3}{4}$ inch near the loop

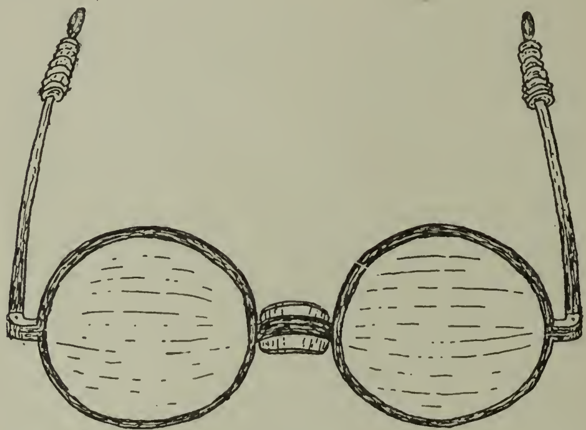

Fig. 10.

should be wrapped with wool. The glasses are tied on by tapes, passed through the loops, behind the child's head. These frames are very comfortable. If the sides were of the usual length the tapes would act at the end of a long lever, causing pressure above the ear, and perhaps ulceration of the tender skin of the infant. When the infant is put down to sleep in the daytime these frames are not removed. These

very short sided do not press on the pillow and lift the glasses from the child's face.

It is usually stated that children under three or four years of age are too young for glasses. No infant is too young to wear glasses should they be required. Many of my squinting patients have begun to wear spectacles such as I have described, long before twelve months of age.

Of course young children sometimes break their glasses, but I have never known a case in which the eye has been injured thereby. The lens, being held in the frame, does not break into splinters but cracks across, or chips at the edge.

(2) Occlusion of the Fixing Eye.—In the case of a child who has squinted constantly with one eye for a considerable proportion of his life, and who has either received no treatment at all or who has been merely given a pair of glasses, one usually finds that the deviating eye has become more or less blind. If the ivory-ball test shows the visual acuity of this eye to be not less than $\frac{6}{36}$, the case may be treated at once in the manner described in paragraph 3. Often, however, one finds that the blindness has progressed far beyond this point, so that the power of central fixation may have been lost and the visual acuity reduced to the ability to count fingers close to the face, or, in some cases, even to bare perception of light.

In a young child, an attempt should always be made to restore, as far as possible, the sight of

the deviating eye, by forcing the child to try to use it. For this purpose I order the fixing eye to be *continuously* occluded for a time. It is not a good plan to order the eye to be occluded for part of each day only; apart from the fact that this is not nearly so rapid and effective as continuous occlusion, the child usually cries every time the shade is applied, so that the treatment is seldom properly carried out. When the better eye is continuously covered, the child soon becomes accustomed to the shade, so that, after a day or two, he usually ceases to object to it.

There are several methods of occluding an eye, each of which may be useful in certain cases. For infants, a gauze pad secured by a few turns of bandage answers well. In the case of children who are beginning to get about, the gauze pad should be held in place by strips of strapping-plaster. Children who are well cared for may have the pad changed every morning. Hospital patients, however, often leave the pad undisturbed for three or four weeks without any harm resulting.

If the child has a reliable nurse in constant attendance it may be sufficient to pack cotton wool behind the spectacle lens. The cotton wool pad must be carefully applied and the child will require constant supervision, or he will push the pad upwards and outwards and peep down the side of his nose. The gauze pad and strapping is

the only possible method for hospital patients, whose parents are seldom able to give them much attention.

I examine the child again at the end of two or three weeks. If the vision of the deviating eye be improved sufficiently, the shade is discontinued and the case treated as described in paragraph 3. If not, the fixing eye is occluded for another month, after which the child is again examined. If occlusion of the fixing eye is going to do much good, one usually finds a very great improvement in the vision of the squinting eye within a fortnight. If this improvement has not taken place at the end of two months it is seldom worth while to continue the shade.

(3) Instillation of Atropine into the Fixing Eye Only.—Atropine has the property of temporarily paralysing the ciliary muscle, and so suspending the power of accommodation of the eye. When, therefore, atropine is instilled into a normal emmetropic eye, or an eye whose refractive error is corrected by glasses, this eye still sees distant objects clearly, but is unable to focus near objects. An unatropised eye whose vision is only one-sixth or even one-tenth of the normal, is able to see objects, at the reading distance, more clearly than a normal eye whose accommodation is paralysed by atropine.

In the case of a young patient, the visual acuity of whose squinting eye has been shown by

the ivory-ball test to be not much less than $\frac{6}{36}$, I order atropine to be put into the *fixing* eye *only* every morning. The child, of course, wears his glasses at the same time. He will be unable to see distinctly his toys and picture-books with the atropised fixing eye. But he quickly discovers that, by making a conjugate lateral movement of both eyes until he has brought the squinting eye to bear on these near objects, he can see them much more clearly. He acquires the habit of always using the (atropised) better eye for distant vision and his (unatropised) worse eye for near vision. In this way the worse eye is most efficiently exercised, and amblyopia ex anopsia prevented. And, in a case in which a considerable degree of blindness has already been acquired, one finds at each visit, if the child be young enough, a steady improvement in vision. In many cases, after a few weeks or months of this treatment, the vision of this previously amblyopic eye becomes perfect, or nearly so. When the visual acuity of the (unatropised) "deviating eye" approaches the normal the child uses this eye always both in near and distant vision, and turns in the (atropised) "fixing eye" instead. I then stop the atropine for two or three weeks to see what will happen. Usually the child returns to his old habit of squinting with the eye with which he squinted at first, and fixing with the "fixing eye." In this case I order atropine every

morning for the *fixing* eye *only* for the first seven days in each month. It occasionally happens, in the case of a young child, that, on discontinuing the atropine, what was originally the fixing eye continues to exhibit the deviation. The case may be left for three or four weeks, but care must be taken lest this originally fixing eye now become amblyopic (see Case B, 83, and Case D, 332, chapter v.). The balance may easily be kept by, if necessary, atropising the other eye for a few days.

The use of atropine for the *fixing* eye *only* is an exceedingly efficient curative measure. A young child spends at least half his waking hours in looking at near objects. So that when he uses the (atropised) "fixing eye" in distant vision and the (unatropised) "squinting eye" in near vision, this is equivalent to perfect occlusion of the "fixing eye" for at least half of each day. And when his glasses are taken off for any purpose, if he has much refractive error, he turns in the atropised "fixing eye" and uses the unatropised worse eye, even in distant vision.

As regards the form in which the atropine is used, I usually order one drop of a 1 per cent. solution of atropine sulphate to be put into the fixing eye only, every morning. It may be inserted with a "dropper" or, better still, with a small camel hair brush or feather. Atropine ointment answers the same purpose. The lower

lid should be drawn down, and a small piece inserted into the conjunctival sac with a glass rod. Lamellæ containing $\frac{1}{200}$ to $\frac{1}{100}$ of a grain of atropine sulphate may be used instead, but nurses usually find them rather difficult to insert.

I always see the child again within a month of beginning this treatment, and after that at intervals of one or two months, according to the nature of the case. In order that there may be no mistake, the mother is given a card on which are written the directions and the date of the next visit. The treatment is continued until the visual acuity of the squinting eye becomes equal to that of the fixing eye, or until no further improvement can be got. Having employed this method for many years, and in a very large number of cases, I am able to state positively that there is no danger of its causing any permanent impairment of the power of accommodation.

This treatment will always prevent the deviating eye from becoming amblyopic. Its efficiency in curing amblyopia which has already been acquired will be greater the younger the child and the more recent the deviation. After about seven years of age usually not much improvement in vision can be obtained, though I have met with many exceptions to this rule.

Of course, carefully keeping the fixing eye tied up for not less than half of the child's waking hours would answer as well, if it could be really

done. But, whatever the mother and nurse may promise at first, they will, after a few weeks, find the amount of supervision required too great a tax on their time and patience. But even the hard-worked mother of a large family, attending a hospital out-patient clinic, finds it no trouble to put a drop of atropine into the fixing eye *only* every morning, for as long as it may be required.

A disastrous practice, adopted by many surgeons and invariably recommended in books, is that of ordering atropine for *both* eyes for children who are supposed to be too young for glasses. The object, of course, is to lessen the convergence by paralysing the accommodation. Atropine, used in this way, never brings about a permanent cure of the squint, though it occasionally causes a temporary suspension of the deviation. But the deviating eye is usually the more ametropic, so that to paralyse its accommodation with atropine is the very way to ensure that this eye shall never under any circumstances be used. The most hopeless cases one sees, of blindness of the squinting eye, are those which have been treated with atropine for both eyes for a few months.

(4) Training the Fusion Sense.—In a case of unilateral or accidentally alternating squint, *if the child is brought early enough*, I endeavour to remove the fundamental cause of the squint by training the fusion sense. For this purpose I use an instrument which I have called "amblyo-

scope." A description of the amblyoscope and of the method of training the fusion faculty will be found in the next chapter. The favourable time for fusion training is between the ages of three and five years. In children under three years of age this treatment is apt to be rather difficult, though I have succeeded in many cases. After five years of age the fusion training takes longer, and a much less powerful "desire for binocular vision" is obtained. After six years of age it is seldom worth while to attempt fusion training at all. It is true that, occasionally, a patient who has squinted for many years may have a sort of binocular vision when his deviation is corrected by operation at a much later date. But this small degree of fusion sense is not a fresh acquisition. He had it before he squinted, but it was too feeble to prevent the occurrence of a deviation, or even to cause diplopia.

Normally the fusion faculty begins to develop at a very early age, and, I think, reaches its full development by about the end of the sixth year. The education of the fusion faculty, at a time when this should normally be developing, is very rapid, easy, and charming in its results. "Stereoscopic exercises," undertaken at a time when the child is old enough to take an intelligent interest in the process, are infinitely tedious and disappointing.

Of course, one should never omit fusion train-

ing in any suitable case which presents itself in private practice. But in a large hospital clinic, it is physically impossible to find time for it except in a few selected cases.

(5) Operation.—In cases of convergent squint in which the deviation is not overcome by other means, operation becomes necessary. Two operative procedures are employed either separately or in combination. They are tenotomy of the internal rectus muscle and advancement of the external rectus muscle.

Tenotomy of the internal rectus consists in a division of the tendon of this muscle at its insertion into the globe. The eye then rotates outwards to an uncertain degree. This outward rotation usually tends to increase as time goes on. The eye also falls forward to a slight extent, so that the tenotomised eye is more prominent than its fellow and its palpebral fissure wider. A permanent weakness of inward rotation (adversion) of this eye is also produced.

In order to *advance* a muscle, the tendon is separated from the globe at its insertion. It is usually then shortened by removal of more or less of the tendon and muscle. The cut end of the muscle is then secured to the globe at a point further forward, nearer the cornea than its original insertion. By a properly performed advancement the eye is rotated to exactly the extent required. The results obtained by the advancement opera-

tion described in chapter xii. are permanent, tending neither to increase nor to decrease with time. Both the power and extent of the rotation of the eye in the direction of action of the advanced muscle are increased. The rotation of the eye in the opposite direction is not in any case weakened in force, though in extreme cases its extent may be slightly diminished. If the abnormal convergence does not exceed 20° or 25°, its cure by advancement of the external rectus muscle rarely causes any noticeable degree of retraction of the globe. If the deviation be of higher degree than this, I usually combine the advancement of the external rectus with tenotomy of the internal rectus, in order to avoid any retraction.

The operations on the external ocular muscles are fully described and discussed in chapter xii.

The question may naturally be asked: "As the deviation in a case of convergent squint is not due to a defect of the muscles, why should one ever seek to remedy this deviation by shortening a muscle?" The answer is this—In the presence of a defect of the fusion faculty, refractive error may cause a deviation, or the equilibrium of the convergence centre may, in some unknown way, be upset by a fright, blow on the head, whooping cough, &c. If one is able to train the fusion sense *early*, the desire for binocular vision is, more often than not, in itself sufficient to overcome the deviation and produce a perfect cure, in

spite of any adverse influences. In many cases, optical correction of refractive error causes the deviation to disappear. In other cases, as one cannot act directly upon the nervous centre which regulates convergence, one has to be content to act upon the peripheral motor organs. Take an illustration—One is driving a pair of horses. Suppose the off-side horse has a habit of boring to the left. If any cause can be found (such as sore shoulder), one may cure the habit by removing this cause. If not, it is a reasonable proceeding to overcome his "deviation" to the left by shortening the off-side branch of the right rein.

The angle of the deviation is measured at each visit. If the measurements show a steady decrease, of course, no operation is indicated under any circumstances. In the case of a young child whose fusion sense I have succeeded in developing by means of the amblyoscope, if the deviation does not decrease at a reasonable rate I have recourse to operation. If the angle of the squint is not higher than 20° or 25°, I advance the rectus externus muscle without tenotomising the internus. If the degree is higher than this, I tenotomise the internus at the same time, not because all the rotation required cannot be produced by advancement alone, but in order to avoid retraction of the globe. This child's eyes having been put approximately straight, his trained fusion sense finds its expression in the act

of binocular vision, and a perfect and permanent cure results.

In a case in which I expect to get binocular vision, I never perform tenotomy of the rectus internus except in combination with advancement of the externus. I once used to do so in hospital practice, occasionally, for want of beds. Some of these patients have since suffered from insufficiency of convergence, and pain in the eyes in near vision. In two or three cases, in which tenotomy was followed by divergence, I have had to advance the tenotomised muscles. It very rarely happens that any evil consequences follow tenotomy of the internal rectus when combined with advancement of the external rectus in high degrees of squint. Perhaps this is because only a small part of the total rotation is produced by the tenotomy, the advancement of the opponent muscle immediately tightening up everything and correcting any tendency to proptosis.

If there is no hope of getting binocular vision, so that cure of the deformity is all that can be accomplished, I prefer not to operate until I am able to do so under cocaine. The eyes are then put exactly "straight" by advancement of the external rectus muscle of the deviating eye, with or without tenotomy of the internal rectus of this eye. One not infrequently sees a patient whose rectus internus has at some previous time been tenotomised, producing proptosis of the eye, but

still leaving some degree of convergent squint. In this case an accurately performed advancement of the external rectus will cure the deviation, and at the same time draw the eye back into its proper position in the orbit.

Simple tenotomy is an unsatisfactory operation, even in cases in which binocular vision is out of the question. The tendon is divided, and the result cannot in any individual case be foretold. Some cases turn out well, others do not. The effect produced by a tenotomy is, on an average, about 13°, though it varies very widely in different cases. The effect, as a rule, increases for the first three or four months after the operation, after which it usually remains about stationary. But in some cases, however skilfully the tenotomy may have been performed, its effect continues to increase for years, until a divergent squint is produced—a more hideous deformity than that which the operation was intended to cure.

There is one condition in which simple tenotomy is sometimes advisable. One occasionally sees a patient whose eyes in distant vision are normally directed, but who turns in one or other eye to an extreme degree in looking at a near object, even in the absence of uncorrected refractive error. One has no means of diminishing the excessive activity of his nervous centre for dynamic convergence, but the case may be treated empirically by tenotomy of an internal rectus.

The internal rectus should never be tenotomised in any case in which dynamic convergence is subnormal.

A patient requires very little care after the operation of tenotomy. This is a great advantage in a crowded hospital clinic. But I think tenotomy owes its popularity chiefly to the extreme facility of its execution, almost no special knowledge being required. An accurately performed advancement, on the other hand, is one of the most delicate (and satisfactory) operations in surgery.

Alternating convergent squint.—The treatment of an accidentally alternating squint is similar to that of a unilateral squint, except that there is no acquired amblyopia to be remedied. It must not be forgotten, however, that such a case, if neglected, may become unilateral, and that the deviating eye may then become amblyopic.

Essentially alternating squints are, fortunately, not very common. The treatment consists in optical correction of any ametropia which may be present, followed, in the majority of cases, by operation. Fusion training is impossible, as there is a total congenital absence of the faculty of acquiring fusion. For this reason, if operation be required, I postpone it until the patient is old enough to permit its performance under a local anæsthetic.

Occasional convergent squint.—The majority of

occasional squints in young children are of the premonitory variety. Optical correction of any refractive error which may be present usually prevents the recurrence of the deviation, and so allows the natural development of the fusion sense to proceed.

Quite half the cases which are commonly supposed to be occasional squints, occurring in older children and adults, are really examples of esophoria (see chapter xi.).

If one whose fusion faculty is perfect suffers from anisometropia of so high a degree as to render the act of binocular vision difficult, he will be likely to manifest an occasional squint. Glasses should be ordered which give the sharpest vision in each eye, even though one eye be hypermetropic and the other myopic. The patient will soon become accustomed to the inequality in the size of the two images. An occasional squint may be caused by hypermetropia in a patient whose fusion sense is feeble.

Vertical deviation.—If operation be required in a case of true vertical deviation, this should consist in advancement of the inferior rectus muscle, or complete central tenotomy of the superior rectus, of the eye which turns up. The inferior rectus should never be tenotomised.

Apparent vertical deviation (p. 37) requires no treatment beyond that of the constant squint.

CHAPTER VIII.

THE METHOD OF TRAINING THE FUSION SENSE.

For more than half a century attempts have, from time to time, been made to teach squinting patients to use both eyes together, by means of exercises with some form of stereoscope. Among the stereoscopes used for this purpose are Wheatstone's original instrument, Brewster's and Helmholtz's stereoscopes, Hering's haploscope, Holme's stereoscope, and, more recently, Javal's "Stéréoscope à cinq mouvements," Priestley Smith's "Heteroscope," Landolt's and Parinaud's stereoscopes, and very many other more or less similar instruments. Most of these are so arranged that they can be adapted to suit the angle of the patient's squint. But the proportion of cases in which they can be used is very small, owing to the suppression of the vision of the patient's deviating eye. Javal attempted to overcome this suppression by prolonged occlusion of one or other eye, in the hope that, when at last both eyes were uncovered, the patient might have diplopia. All these instruments are intended for the use of patients who are old enough to intelligently follow the directions given them.

Probably most ophthalmic surgeons have, at some time or other, been in the habit of ordering "stereoscopic exercises" in cases of convergent squint, and have, after careful trial, given them up as useless. The reason of their failure is the very early age at which the fusion sense normally develops. Fusion training, to be of any material benefit, must be undertaken within this normal period of development.

Fusion training of young squinters under five years of age is in suitable cases, quickly and easily accomplished, and the results obtained are most striking and gratifying. Between five and six the treatment is apt to take longer, and the results to be less perfect. After the age of six or, at the latest, seven years, the results are, from the patient's point of view, not worth the time and trouble which they cost.

There are two great difficulties in the way of fusion training in the case of young children :—

(1) Though the visual acuity of the child's deviating eye may perhaps be perfect, the vision of this eye is suppressed, so that he is ordinarily unable to receive impressions from it except when the other eye is closed.

(2) The child is far too young to understand the purpose of fusion training or to follow the directions of the surgeon. He will, therefore, only permit the exercises so long as he finds them attractive and interesting.

After many experiments, I devised an instrument with the help of which I have to a great extent succeeded in overcoming these difficulties. I have called it "amblyoscope"—an instrument by means of which a non-seeing eye is trained to take its share in vision. The amblyoscope has retained its present form since 1895.

The Amblyoscope.[1]—The instrument consists of two halves joined together by a hinge. Each half consists of a very short brass tube joined to a longer tube at an angle of 120°. At the angle of junction of the tubes is an oval mirror,[2] protected on the outside by an oval plate of brass. Each half of the instrument has at its distal end an object-slide carrier, and at its proximal end a convex lens having a focal length of five inches—the distance of the reflected image of the object-slide. In front of each lens is a slot into which a prism, axis vertical, may be inserted if required. The diameter of the tubes is $1\frac{1}{2}$ inch.

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[1] The amblyoscope is made by Mr. Hawes, optician, 79, Leadenhall Street, London, E.C., and by the principal manufacturing opticians in England and abroad.

[2] The mirrors must be extremely thin. If they are merely pieces of ordinary thick mirror glass there will be blurring, owing to reflection from the surface of the glass as well as from that of the mercury. Mirrors silvered on the surface answer well but are easily scratched. I have tried polished speculum metal. This is satisfactory but very expensive. The mirrors must be accurately set perpendicular to the plane of the tubes.

A brass arc[1] connects the two parts of the instrument, being clamped on one side by a binding screw set in a long slot and on the other by a binding screw set in a short slot. When the screw in the long slot is loosened, the two parts of the instrument can be brought together to suit a con-

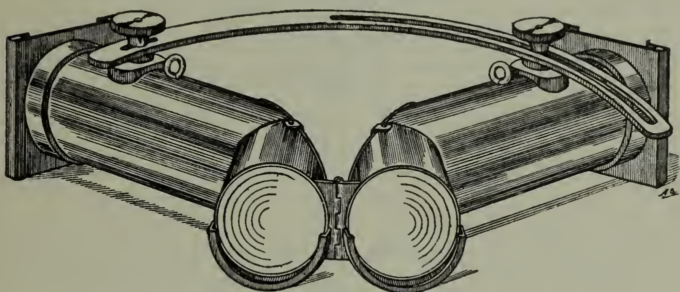

FIG. 11.

vergence of the visual axes up to 60°, or separated to suit a divergence of as much as 30°. When this screw is tightened and the screw in the short slot loosened, an amplitude of movement of about 10° only is permitted.

The convex lenses of course render unnecessary any adjustment of the instrument for the patient's inter-pupillary width.

[1] In the earlier forms of the instrument I had the arc marked in degrees, as I thought that the instrument might also be used for the subjective measurement of heterophorias. I found, however, that it was of no use for this purpose. No instrument, in which the objects looked at are near the eyes, is reliable for measuring heterophoria. Though the lenses render accommodation unnecessary, the patient unconsciously accommodates for an object which he knows is near.

Illumination of the object-slides.—Each object-slide is illuminated by a separate electric lamp.

A stout brass rod, about two feet in length, is held in brass sockets at each end. The brass sockets are screwed to a board, which is firmly

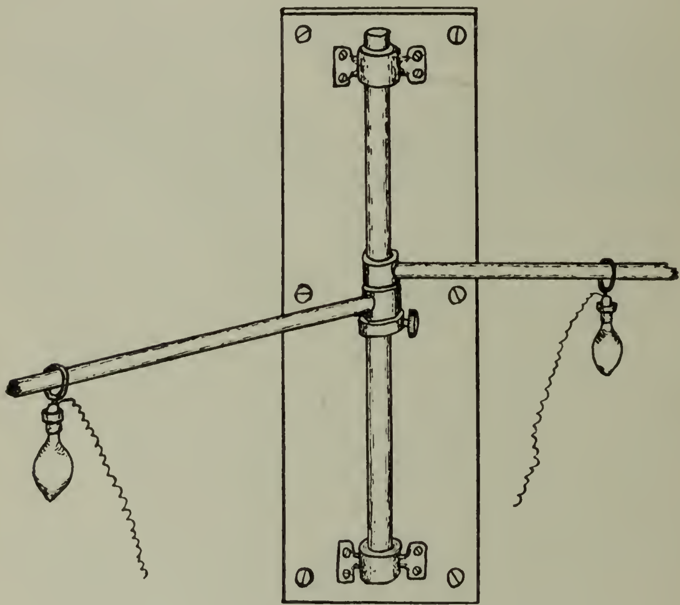

FIG. 12.

secured to the wall of the consulting room in such a position that the brass rod is vertical. A brass collar slides up and down the vertical rod, and is fixed by a thumb-screw at the height required. This collar supports two independent collars, each of which has attached to it a long horizontal arm. Each of these arms is four feet in length, and is

free to move in a horizontal plane independently of the other. An electric lamp is suspended under each horizontal arm from a ring which slides along the arm. The illumination of either of the object slides in the amblyoscope may be separately increased or diminished by bringing its lamp nearer or pushing it further away.

Before I had electric light I used two paraffin lamps on a table, varying the distance of each lamp and the height of the flame as required. This simple plan answers just as well but is not quite so convenient.

The object-slides.—Fig. 13 shows the familiar vertical slits with their control marks. They are not used for fusion training, one could not induce a young child to study such uninteresting objects. They are shown because they are useful in making experiments with older persons.

The devices used in fusion training are of three classes :—

(1) Those which do not require any blending of images, but only simultaneous vision of dissimilar objects with the two eyes. Fig. 14, showing a cage on one slide and a bird on the other, is an example. Other pairs of devices of this class are a clown and a hoop, a mouse and a trap, a clock-face and hands, &c.

(2) Devices of the second class, of which figs. 16 and 17 are examples, require true fusion of images in order that the full picture may be seen.

FIG. 13.

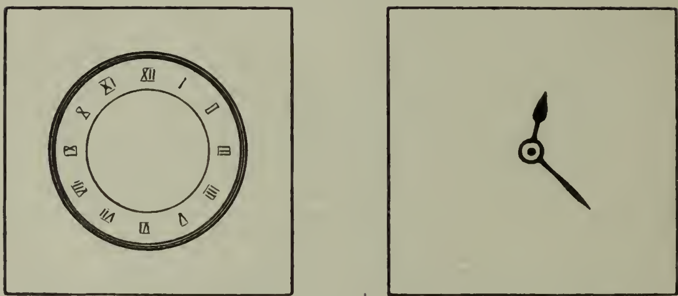

FIG. 14.

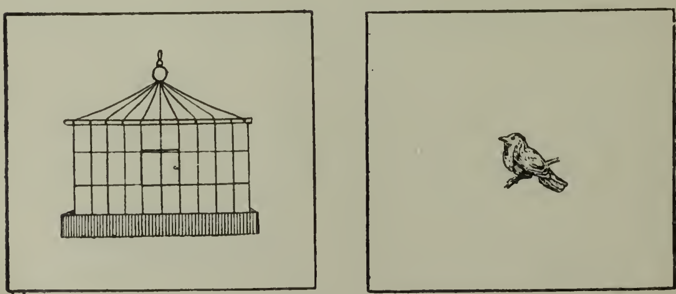

FIG. 15.

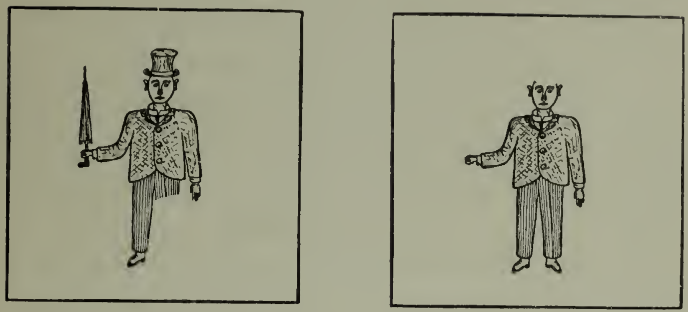

FIG. 16.

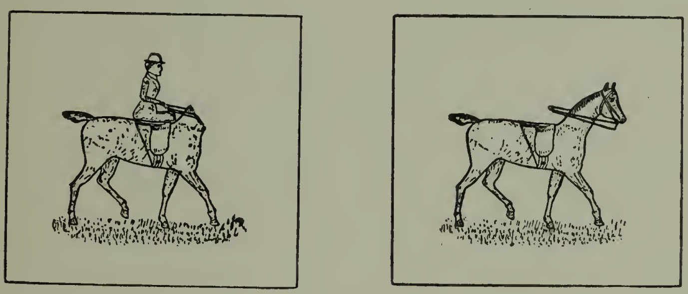

FIG. 17.

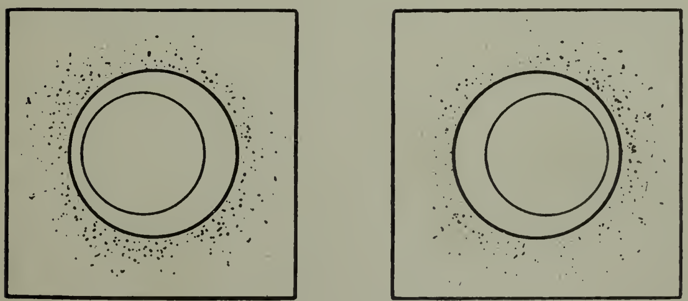

FIG. 18.

The pictures on each pair of slides are exactly similar, except that part of the design is omitted in one and a different part in another. For example, in fig 16 a leg is omitted in one slide and the hat in the other. A child who blends the images sees a man with two legs and a hat.

(3) Devices such as fig. 18 can only be appreciated by patients who have the third grade of binocular vision, the sense of perspective.

The designs are drawn on $1\frac{1}{2}$ inch squares of thin translucent paper. The paper is then pasted on squares of glass. There is no difficulty in making designs of the first class. Object slides of the second class are made as follows :—I draw, on pieces of paper $1\frac{1}{2}$ inch square, rough, simple pictures, such as a horse, a clown, a cow, a man with a hat and pipe, &c., the more grotesque the better the children seem to like them. I then make two tracings of each on the translucent paper, omitting a different part of the picture in each tracing.[1]

Fusion Training.—The education of the fusion

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[1] "Stereoscopic views" are of no practical use whatever for fusion training. But I once had made some tiny transparent stereoscopic photographs to fit the amblyoscope. My idea was that a child who wearied of my own artistic efforts might be shown some of these views, occasionally, by way of a change. But I found that young children took no interest in them. They much preferred simple pictures which they could understand.

sense should be undertaken at the earliest possible age. I repeat this because it is of supreme importance—the only key to success. In the case of a child of average intelligence, it is quite easy to use the amblyoscope at the age of three to three and a half years. I have succeeded in many cases before three years of age, but it is often rather difficult to keep the attention of these very young children.

The deviating eye must not be too blind. In cases which have received *efficient* treatment since soon after the first appearance of the deviation, the vision of each eye is nearly always perfect. But, in neglected or inefficiently treated cases, the deviating eye is often very blind. (Compare Tables III. and V., pp. 76 and 77). I do not as a rule attempt fusion-training in a child who, after all possible means have been employed to restore the sight of the deviating eye, is still unable to see the 1 inch ivory ball at six yards.

A squinter sees with his fixing eye only, or, under certain circumstances, with the deviating eye only, but not with both simultaneously. The first step in the treatment is to overcome this suppression. If the child has any refractive error this is corrected by spectacles. It occasionally happens that the mirror test (p. 80) has revealed a slight vertical deviation. In this case corresponding prisms, axes vertical, are inserted in the slots in front of the lenses. Seated in a chair facing the

lighting apparatus, I take the child on my knee and adapt the amblyoscope[1] roughly to the angle of his squint. I put in the instrument first a pair of object-slides which requires no fusion, only simultaneous perception—those shown in fig. 15, for example. Each light is at first about 4 feet away from its object slide. Suppose the cage is before the fixing eye and the bird before the squinting eye. The child, on looking into the amblyoscope, will see only the cage. I tell him to look for the bird, while I bring the light before the deviating eye nearer and nearer. At last a point is reached when the illumination of the object-slide before the deviating eye becomes so intense that the vision of this eye can no longer be suppressed. The child suddenly says he sees the bird he was told to look for. But he has now usually lost sight of the cage before the fixing eye. The relative distances of the lights are then readjusted until, after a few minutes of alternation of the vision of the two eyes, the child sees the bird and the cage simultaneously. The child is then allowed to grasp the amblyoscope

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[1] I used occasionally to drop the instrument and break some part of it, until Dr. Ernest Maddox suggested the simple expedient of slinging it from the ceiling by means of a string and pulley and little leaden counterpoise. Since then there have been no mishaps. When pulled down ready for use the amblyoscope hangs about 3ft. away from the wall-bracket which supports the lighting apparatus.

with both hands, while I, putting my hands over his, converge and diverge the two halves of the instrument, in order to make the bird appear to go in and out of the cage. Other similar pairs of object-slides are then shown. The child soon learns to move the instrument himself, so as to put the bird in the cage, the cat on the chair, the clown in the hoop, &c. One must talk to the child all this time, as it is only by encouraging him to chatter that one learns what he really sees.

A pair of slides requiring fusion of images is now shown—fig. 16, for example. The child at first sees two men, each picture being imperfect. Soon a position is found in which the child sees one man having two legs and a hat. The binding screw in the long slot is now tightened and that in the short slot loosened, so that the amplitude of movement between the two halves of the amblyoscope is restricted to about 10°. Other pairs of slides requiring fusion of images are successively shown the child, and he is encouraged by one's remarks to examine every part of the fused picture. After a time it is found that the angle of convergence of the instrument may be varied slightly without the fused picture coming to pieces. The child has now, under these special conditions of illumination and convergence, the second grade of binocular vision, —true fusion with some amplitude.

The next step is to increase the amplitude of

fusion. The intensities of the lights and the angle of convergence of the amblyoscope are arranged as before. Devices requiring fusion, such as figs. 16 and 17, are shown. An attempt is made to gradually diverge and converge the two halves of the instrument, more and more, while the child is examining and talking about the various pictures shown him. After some practice, in the case of a young child, a considerable range of movement becomes possible, fusion being still maintained. This "amplitude of fusion" may, for practical purposes, be taken as a measure of the extent to which the fusion faculty has been developed.

A child who has any considerable amplitude of fusion will nearly always be found to have acquired the third grade of binocular vision also—the sense of perspective. The slides, fig. 18, are shown, and the child is asked whether he is looking at the outside or the inside of the tub. He will at once say "the inside." If these slides are now changed from one tube to the other, he will see the tub bottom up.

The child having now acquired the highest grade of binocular vision under these special conditions of illumination and convergence, the next step is to gradually equalise the light before the two eyes. This may readily be done, at this stage, without a return of the suppression.

If the child be young enough, a very powerful

"desire for fusion" may usually be created in five or six lessons, given at intervals of one week.

It must be remembered that the prime object of these exercises is the training of the fusion sense at a time when this may be successfully accomplished—not a mere remedying of the deviation. In many cases, however, the powerful "desire for fusion" thus established, directly brings about a sudden cure of the squint. In the larger group of cases, in which optical correction of refractive error is depended upon to lessen, and perhaps overcome, the deviation, there is no danger of the newly-acquired faculty of fusion being lost meanwhile. If even a faint degree of fusion sense has once been acquired, its persistence is truly remarkable. No matter how many years binocular vision may be in abeyance, it seems as difficult to forget as the art of swimming. If the periodic measurements of the angle of the deviation show that this is not decreasing reasonably quickly under optical treatment, I have no hesitation in operating at any age, by advancement of an external rectus, with or without tenotomy of the rectus internus. The eye being put approximately "straight," the "desire" for binocular vision fills up any slight gap that may remain, and a perfect cure results.

Fusion training, in the case of these young children, must be carried out by the surgeon himself. It is of no use giving the amblyoscope, or

any other instrument, to the mother for use at home.

In the case of older children, I have often, by way of experiment, ordered some form of stereoscope, or Cuignet's (or Javal's) "lecture contrôlée" for home use. I have not, in any single instance, seen any good result therefrom. A child who is old enough for "stereoscopic exercises" and "bar reading" is far past the age when the fusion sense might have been developed. Similarly, if he has suffered from neglected constant unilateral squint since infancy, it will now almost certainly be impossible to remove the acquired amblyopia of the squinting eye, though it might easily have been done at an earlier age.

In this description of fusion training I wish to draw attention rather to the principles than to the instruments by the help of which they have been defined and applied. Though these methods have been constantly employed for many years, it is possible that they may be improved upon; but the principles are unalterable.

CHAPTER IX.

DIVERGENT SQUINT.

Comitant divergent squint presents two distinct varieties differing widely in their pathology, appropriate treatment and prognosis. They may be called, respectively, myopic and neuropathic.

Myopic Divergent Squint.

The divergence most frequently first makes its appearance at about ten or twelve years of age. It may be either unilateral or alternating, more commonly the former. Usually the patient is known to have been short-sighted for four or five years, and the short-sightedness has been increasing, until, at the time of the appearance of the divergence, there is myopia of perhaps 5 or 6 D. or more. As a rule the fusion sense is well-developed, though I have sometimes found it deficient. The deviation is seldom constant; at one moment the eyes may be "straight" and the patient have binocular vision; a few minutes later one or other eye may exhibit a high degree of divergence. Myopic divergent squints of low degree are rare.

From observing and carefully questioning many of the more intelligent patients, I conclude that the usual mode of origin of a myopic divergent squint is as follows :—Soon after the child begins school it is found that he is not easily able to see the black-board. He is given a front seat where he can see, and nothing more is done. A few years later, say at twelve or thirteen years of age. the myopia has increased so as to bring his far point very near his eyes. All distant objects are blurred. In reading, he has to hold his book so near his eyes that it becomes very difficult to converge steadily to the required extent. He complains that "the words run into each other" and he soon tires. One frequently hears that, at about this time, he discovered that, by covering one eye with his hand, he could read quite comfortably with the other. In any case, he gives up the struggle and allows one eye to wander outwards, while he reads without effort with the other. At first there is no actual divergence—only a failure to converge while the other eye is engaged in near vision. But, as a result of convergence not being used, the function becomes weakened, so that either eye diverges when screened or when the other is being used in near vision. When the myopic eye has become divergent there is no diplopia in reading, even though the fusion faculty be perfect, because the divergent eye is directed towards the misty distance. When the patient

looks up from his book, the divergent eye at first always recovers itself. After a time, as the habit becomes confirmed, this eye often remains widely divergent in distant vision. The picture of distant objects formed in the fixing eye is already blurred and indistinct, so the very faint eccentrically-placed image formed in the divergent eye causes no tendency to fusion. But when the divergent eye partially recovers its normal direction, so that it receives a more centrally-placed image of the object to which the fixing eye is directed, the desire to blend these two blurred images causes the eyes to become straight. This probably explains why in a case of myopic divergent squint, the eyes are sometimes straight, sometimes widely divergent, but scarcely ever divergent to a slight degree.

The *treatment* of a case of myopic divergent squint consists in exactly correcting the myopia and myopic astigmatism. The patient should wear the glasses always, both for near and distant vision. Patients at first complain that the glasses make print appear very small. Children soon become accustomed to them. But myopes who first begin to wear glasses in adult life, in some cases, require a second pair of glasses for near vision, 1 D. or 2 D. weaker than the distance glasses. Their power of accommodation has become feeble, for want of exercise. Glasses with which the patient can see distinctly usually

cause a rapid disappearance of the squint, in fairly recent cases, and frequently even in cases which have lasted for many years. But it often happens that the prolonged disuse of the function of dynamic convergence weakens the static convergence, so that this becomes a negative quantity, and a tendency to divergence remains. While both eyes are open and the glasses are worn, the fusion faculty prevents any deviation, but if one eye be shaded for a moment it may diverge and remain divergent for a second or two after the removal of the shade. Operation is rarely necessary. But in some cases, usually of long standing, in which the static convergence remains very deficient, advancement of an internal rectus adds greatly to the patient's comfort.

It must not be forgotten that the case may be complicated by muscular exophoria.

Infantile myopic divergent squint, excluding cases of buphthalmos, is very rare. I have notes of only seven cases. These cases differed from unilateral convergent squints only in the kind of refractive error and the direction of the deviation. The treatment was similar to that employed in convergent squint.

Neuropathic Divergent Squint.

The divergence nearly always dates from infancy. It may be constant or occasional, unilateral or alternating. In the constant cases there is a

total absence of the fusion sense. In the occasional cases there is a feeble degree of binocular vision when the eyes are "straight"; when an eye diverges there is usually no diplopia. The refraction, as a rule, is normal. In the constant unilateral cases there may be acquired amblyopia. In other cases the vision of the eye is perfect. The divergence varies greatly in degree, even in a case in which it is constantly present. The power of dynamic convergence is deficient. It varies from time to time in an extraordinary manner. One day, perhaps, the dynamic convergence may be almost normal; a few days later the most persevering efforts may fail to elicit the slightest movement of convergence. In these neuropathic divergent squints, the association between accommodation and convergence is usually very slight. One not infrequently sees a patient who can nearly always voluntarily correct the faulty position of his divergent eye without much effort, but who habitually allows this eye to diverge while he is exercising say 4 or 5 D. of accommodation in near vision. In young subjects the power of rotation of each eye separately is nearly always normal in every direction. In long-standing cases, however, the power of independent inward rotation is usually deficient.

The subjects of neuropathic divergence are often bright, quick-witted, intelligent, but they are nearly always very "nervous" and highly strung.

One frequently sees the same ocular defect in other members of the family. A family history of epilepsy or insanity is very common. I find neuropathic divergence more common in females than in males, in the proportion of about 7 to 2.

The *treatment* of neuropathic divergent squint is not satisfactory. There is seldom any notable refractive error, and when there is, its correction produces no effect upon the divergence. Attempts at fusion training nearly always fail, however early the patient may be seen. Practically one's only recourse is operation. One or both internal recti should be advanced (*e.g.*, Case B, 166, p. 162). The externi should be tenotomised at the same time only when the power of abversion is distinctly greater than normal. If the power of adversion is very markedly deficient, a musculo-capsular advancement should be performed. In a case in which divergence is not constantly present and in which there is some slight degree of fusion, advancement gives a satisfactory result (*e.g.*, Case B, 165, p. 161).

Non-comitant Divergent Squints, other than Paralytic.

Divergence in extreme myopia.—In cases of progressive myopia of very high degree it is not uncommon to find the visual axes divergent. These cases differ entirely from ordinary myopic divergent squints. They are non-comitant. The

arcs of rotation of each eye are subnormal in every direction. The divergence begins imperceptibly, and increases slowly up to about 20°. It is generally held that the divergence is caused mechanically, by the egg-shaped eyes adapting their long axes to the divergent positions of the orbits. I believe this explanation to be correct. Increased tension of the external recti does not appear to be an important factor, as I have, in three such cases, seen both these muscles tenotomised without result.

Divergence of blind eyes. – When both eyes are blind they almost invariably diverge. When one eye only is blind, its behaviour will depend, to a great extent, upon the state of the refraction of the seeing eye. If this is normal or myopic the blind eye will, as a rule, diverge; if it is markedly hypermetropic it will usually converge.

If the refraction of one eye be normal, or nearly so, while the other eye is very highly myopic, the latter is, in the absence of optical correction, practically a blind eye, and will behave as such.

In a case of convergent squint in which the squinting eye has become very amblyopic, this blind eye may become divergent in after years, without any tenotomy having been performed.

Divergence secondary to tenotomy of an internal rectus muscle.—As already explained, in a large proportion of cases of convergent squint with hypermetropia, the constant wearing of glasses

gradually causes the visual axes to become less convergent. It was formerly the practice to tenotomise an internal rectus in almost all cases of convergent squint, without going into the question of refractive error. Such of the hypermetropes who subsequently took to wearing convex glasses to improve their vision, ran considerable risk of divergence.

If tenotomy be performed only in cases in which optical treatment has failed, the risk is less. But even then a case of divergence will occasionally be seen. In an ordinary tenotomy the tendon and its lateral expansions are divided. Usually the cut end of the tendon becomes connected to its old insertion by an irregular band of scar tissue, which may or may not subsequently stretch. Tenotomy is called a "setting back" of the tendon, because it is supposed that the tendon becomes directly reattached to the globe further back. I believe that this seldom happens after a neatly-performed tenotomy. Sometimes the tendon fails to become reattached to the globe at all. In this case the surgeon may fail to find the muscle unless he knows where to look for it. Its anterior end will be found behind the sunken caruncle, lying against the inner wall of the orbit (see page 213).

CHAPTER X.

THE TREATMENT OF SQUINT. ILLUSTRATIVE CASES.

The following clinical notes, copied from my squint case-books, will serve to further explain the methods of treatment which I employ, and to show the results which may ordinarily be expected therefrom.

As a rule, it is only possible to get a perfect result in a case in which efficient treatment is commenced early. But, unfortunately, one sees a very large proportion of the cases for the first time after years of neglect, or perhaps inadequate or even harmful treatment. I have, therefore, also selected some examples of these old cases.

Case B, 23. *February* 4, 1896.—A boy, aged 2 years 11 months, was brought to me, suffering from convergent squint. He had squinted for about ten or twelve months. L. E. was convergent 28°. Fixation was present in L. E., but the sight of this eye had deteriorated so much that, when R. E. was bandaged, he could not find a white-handled penknife on the floor, unless it was close to his feet. Abversion L. E. was perfect. Ordered, atropine ointment, 1 per cent., thrice daily for both eyes, for retinoscopy.

February 11.—Convergent squint L. E. 32° with

atropine. Retinoscopy—each eye + 2·75 D. sph. + 0·5 D. cyl. ax. vert. Ordered spectacles + 2·25 D. sph. + 0·5 D. cyl. ax. vert., also guttæ atropinæ 1 per cent. for R. E. *only* every morning.

March 3.—Child uses R. E. (atropised) in distant vision, and L. E. (unatropised) in near vision. Convergent squint L. E. 20°, with glasses. Ordered, continue.

April 9.—Similar note. Convergent squint L. E. 17°. Ordered, continue.

May 29.—Child now uses (unatropised) L. E. and turns in (atropised) R. E. always, both in near and distant vision. Ordered, stop the drops, and come again in one month.

June 18.—Squint nearly alternating, slight preference for squinting with L. E. Convergent squint L. E. 16°. Ordered, atropine for R. E. only, every morning for a month, then stop it and bring the child to me two or three weeks later.

August 4.—C. S. alternating 14°. Fusion training with amblyoscope. Child sees both images readily and can sometimes blend them.

August 7.—Fusion training.

August 10.—Fusion training. Child blends images readily.

August 14.—Fusion training. Child has considerable amplitude of fusion. C. S. alternating 15°.

August 18.—Fusion training.

August 25.—No squint with glasses. The mother says that the eyes have been straight since the last lesson, though he has turned the eye in, occasionally, for an instant.

November 6.—Child never squints now with glasses, though he occasionally does so without them, the nurse says. No squint seen now either with or without glasses.

October 8, 1897.—Child going on well. He never squints now.

September 29, 1898.—Spectacles too small. Ordered, repeat them.

June 13, 1901.—(Aged 8 years 3 months). Ordered, atropine for repetition of the retinoscopy.

June 20.—Retinoscopy each eye + 2·25 D. sph. + 0·5 D. cyl. ax. vert. Vision, each eye $\frac{6}{6}$. Ordered, spectacles 0·5 D. lower than the retinoscopy, with permission to take them off while playing games.

August 19, 1902.—The boy has only used his glasses for school work since last visit, and never when out of doors. He has not been seen to squint for years.

CASE B, 18. *November* 7, 1895.—A girl, aged 2 years 1 month, was brought to me, suffering from C. S. R. E. 33°. She had squinted since she had whooping cough aged 1 year 4 months. She had central fixation in R. E., but she evidently did not see well with it when the L. E. was tied up. Abversion perfect. Bright, intelligent girl, only child. Mother has C. S. R. E. Ordered, atropine for retinoscopy.

November 13.—Retinoscopy R. E. + 1·75 D. sph. L. E. + 1·25 D. sph. C. S. R. E. 26°, with atropine. Ordered, spectacles 0·25 D. less than retinoscopy; also guttæ atropinæ 1 per cent., one drop in L. E. *only*, every morning.

December 11.—Child uses R. E. (unatropised) in near vision, and L. E. (atropised) in distant vision. Ordered, continue.

February 6, 1896.—Same. Ordered, continue.

April 2.—Child uses R. E. (unatropised) now, both in near and distant vision, and squints with atropised L. E. C. S. L. E. 27°. Ordered, stop atropine.

June 4.—Squint alternates 22°. Fusion training with amblyoscope: child readily sees both images, but I cannot be certain that she blends them.

June 10.—Fusion training: child learned to blend images quite easily.

June 19.—Fusion training.

July 1.—Fusion training.

July 7.—Fusion training. Child now has an amplitude of fusion, with the amblyoscope, of 10° to 15°. C. S. alternating 23°. As the fusion faculty is well developed and the deviation is stationary, I advised operation, especially as, in view of the insignificance of the refractive error, spectacles can be dispensed with. The parents preferred to wait until the child was 3 years old. Child's fusion was exercised with the amblyoscope once a month until

October 5 (aged 3 years).—C. S. alternating 22°. I advanced the right external rectus muscle, under chloroform. (I employed the method described in Chapter XII.).

Case dressed every day.

October 12. Sutures removed.

November 3.—R. E. is not opened quite so widely as L. E., and there is still some redness of the conjunctiva. There is no squint now, and the child has perfect binocular vision. She, of course, has not worn her glasses since the operation.

I saw the little girl in January, 1897, October, 1897, June, 1899, and again on

November 12, 1902.—I wrote and asked the mother to bring her to me. The child is now nine years old. She has $\frac{6}{6}$ vision, each eye, and perfect binocular vision. She only dimly recollects that she ever had anything the matter with her eyes.

CASE B. 192. *November* 20, 1899.—A lady, aged 23 years. History—she began to squint with the L. E. when she was about 1 year old. She was treated from time to time with "drops." At about 4 years of age she was ordered glasses. She has worn glasses ever since. When she was 8 years old the L. E., was operated upon (tenotomy).

She now has convergent squint L. E. 16° both with and without her glasses. Vision of R. E., with glasses, is $\frac{6}{6}$. L. E. has lost central fixation, and with it she can just count fingers close to her face. As the result of the tenotomy, the L. E. is prominent and the caruncle sunken. Retinoscopy under homatropine—R. E. + 1 D. cyl. ax. vert., L. E. + 1·75 D. cyl. ax. 70° down and out, at approximate macula. The blindness of the left eye is now, of course, quite incurable. She wishes to have something done to remove the deformity.

December 5.—I advanced the left external rectus muscle, under cocaine (by the method described in Chapter XII.).

December 20.—There is a little redness remaining from the operation. The eyes are quite "straight," and the advancement has had the effect of replacing the L. E. in its proper position in the orbit. Ordered, rigid pince-nez + 1 D. cyl. ax. vert.

June 14, 1901.—Eyes quite normal in appearance, both with and without the glasses.

CASE D, 318. *April* 13, 1900.—Boy, aged 1 year 5 months, seen at Moorfields Hospital. The mother said he had measles in February and he "has squinted the last few weeks." C. S. R. E. 37°. Steady central fixation R. E. Ordered atropine, for retinoscopy.

April 24.—C. S. R. E. 32° under atropine. Retinoscopy R. E. + 5·5 D. sph. L. E. + 4·5 D. sph. Ordered, spectacles R. E. + 5 D. L. E. + 4 D. Guttæ atropinæ 1 per cent. to be put in L. E. *only* every morning.

May 8.—Child uses (unatropised) R. E. and squints (atropised) L. E. always. Ordered, stop drops.

June 5.—Squint alternates, 21°.

August 7.—No squint while wearing glasses. C. S. R. E. about 20° without glasses.

September 10, 1901.—Glasses too small now, repeat them.

November 7, 1902.—No squint seen here either with or without glasses. He is said to turn in R. E. sometimes when his glasses are taken off at bedtime.

CASE A., 489. *May* 10, 1900.—Boy, aged 3 years 4 months, seen at the West Ham Hospital. C. S. L. E. 34°. Fixation L. E. was lost. Abversion L. E. full. The mother said he had "squinted on and off for about a year." Ordered, atropine for retinoscopy.

May 17.—C. S. L. E. 27° with atropine. Retinoscopy—R. E. + 3·75 D. sph. + 0·75 D. cyl. ax. 25° down and out. L. E. approximately the same. Ordered, spectacles for constant wear, R. E. + 3·5 D. sph. + 0·75 D. cyl. ax. 25° down and out. L. E. + 3·5 D. sph. *pro tem.*: also R. E. to be occluded by a gauze pad secured by strapping plaster, for one month.

June 14.—Steady central fixation L. E. Ordered, guttæ atropinæ 1 per cent. for R. E. *only* every morning for one month.

July 12.—Child now uses (atropised) R. E. in distant vision, and turns in L. E. In near vision he uses (unatropised) L. E., and turns in R. E. C. S. L. E. 23°. Ordered, continue.

August 30.—Child uses R. E. distant vision and L. E. near vision. When glasses are taken off, he always fixes with L. E. and turns in the (atropised) R. E. Ordered, continue.

October 25.—Child uses (unatropised) L. E. and turns in the (atropised) R. E. always, both with and without the glasses. Ordered, stop the atropine and return in one month.

December 6.—C. S. L. E. 10°. The child uses L. E. almost as readily as R. E. Fusion training with amblyoscope.

December 13.—Fusion training.

December 20.—Fusion training. Child blends images, but with difficulty.

January 10, 1901.—Fusion training.

January 24.—Fusion training. Images blended at once. A fair amplitude of fusion.

February 7.—No squint with glasses.

July 11.—No squint with glasses. When glasses are taken off the child has no squint, as a rule; but when he is told to look at a picture L. E. turns in about 35° to 40°, and he says he sees two books (he volunteered this statement without any questioning).

May 29, 1902.—Child never squints now. Ordered, atropine for both eyes for repetition of the retinoscopy.

June 7.—Retinoscopy, R. E. + 3·5 D. sph. + 0·75 D. cyl. ax. 30° down and out. L. E. + 3·5 D. sph. + 1 D. cyl. ax. 30° down and out. Vision with these glasses R. E. $\frac{6}{6}$, L. E. $\frac{6}{9}$. Ordered, spectacles 0·5 D. less than the retinoscopy.

Case D, 832. *May* 6, 1902.—A boy, aged 9½ years, was brought to the Royal London Ophthalmic Hospital, because he could not see with the right eye. The father said that the boy squinted when he was teething. He was taken to an eye hospital where he was given ointment (probably atropine) for both eyes for about a year. At about 3½ years of age he was given glasses. The glasses gradually "cured the squint but the sight of the eye, which used to squint, is almost gone."

The boy is wearing + 4 D. sph. each eye. Vision with his glasses—R. E. counts fingers at two feet, L. E. $\frac{6}{9}$. Fixation R. E. is, of course, lost. He has C. S. R. E. 4°, with his glasses. Abversion R. E. full. Ordered, atropine for retinoscopy.

May 9.—Retinoscopy L. E. + 4 D. sph. + 1 D. cyl. ax. vert. R. E. approximately the same. Vision with glasses—R. E. not improved, L. E. $\frac{6}{6}$. Ordered, spectacles 0·5 D. less than retinoscopy. The blindness is now of so long standing that it would be quite hopeless to attempt to restore the sight of the R. E.

Case A, 58. *April* 23, 1894. I saw a girl, aged

2 years 3 months. Alternating C. S. about 30°. She had squinted since early infancy.

April 26, 1894.—Retinoscopy under atropine.—Each eye + 1·5 D. sph. Ordered, + 1·25 D. sph.

November 7, 1895.—C. S. alternating 32°. Attempted fusion training with amblyoscope.

During November, 1895, I made six attempts to train the child's fusion sense. Though she was a tractable and intelligent child, I did not succeed in getting even simultaneous perception of the object slides. She could see one picture or the other, but she could not see both together. (Case of essentially alternating squint, with total absence of the fusion sense.)

August, 1900.—The girl came to me at the West Ham Hospital. She has not worn the glasses for two or three years. Two years ago she went to another hospital, where the right internal rectus muscle was tenotomised. C. S. alternating 19° now. Vision $\frac{6}{6}$ each eye.

October, 1900. I advanced the right external rectus muscle, under cocaine.

November 8, 1900.—The eyes are "straight" and normal in appearance. The vision of each eye separately is perfect. But of course she can never have binocular vision. No glasses required.

June 12, 1902.—Eyes normal in appearance, patient comfortable.

Case B, 3.—*May* 22, 1893. I saw a girl, aged 1 year 5 months. She had C. S. R. E. about 35°. She had squinted for six or seven months. R. E. has not lost the power of central fixation. Ordered, atropine for retinoscopy.

May 25.—Retinoscopy each eye + 4 D. sph. Ordered, glasses each eye + 3·5 D. sph.: also, guttæ atropinæ 1 per cent. L. E. *only* every morning.

June 26, 1893.—Glasses worn well. When the child

is induced to look at anything close to the eyes, she uses the R. E. (unatropised) and turns in the (atropised) L. E. At other times she has C. S. R. E. about 30°. Ordered, continue drops for L. E. *only*.

July 28.—Child uses L. E. (atropised) in distant vision and R. E. (unatropised) in near vision. When glasses are taken off, she always uses R. E. and squints with (atropised) L. E. Ordered, continue.

September 5.—Child always uses R. E. and squints with the (atropised) L. E. both in near and distant vision. Ordered, stop the atropine.

April 23, 1894.—C. S. nearly alternating. More often fixes with the R. E., which was at first the squinting eye. C. S. 21°.

November 6th, 1895.—Owing to my absence abroad the child was left much longer than she should have been. C. S. L. E. 13°. Ordered, atropine for repetition of retinoscopy.

November 13. Retinoscopy + 3·5 D. sph. + 0·5 D. cyl. ax. vert. each eye. Ordered, glasses 0·5 D. less than the retinoscopy. As the child now squinted constantly with L. E. (the originally fixing eye) I ordered atropine to be put in the R. E. *only* every morning.

November 27.—She uses R. E. (atropised) in distant vision and L. E. (unatropised) in near vision. Ordered, continue.

December 20.—Child now uses L. E. almost always in preference to the (atropised) R. E. Ordered, stop atropine.

January 15, 1896.—C. S. nearly alternating 11°. Fusion training.

January 20.—Fusion training.

January 27.—Fusion training. Child blends images.

February 4.—Fusion training. Child has some amplitude of fusion.

February 13.—Fusion training. Considerable amplitude of fusion.

February 20.—Child has frequently said that she sees two faces, &c., since last visit. Fusion training.

February 27.—No squint.

June 5.—No squint with glasses. C. S., with diplopia, when glasses are taken off. Diplopia is so intense that the little girl remarks it at once.

October 19, 1898.—Glasses too small. Ordered, repeat the glasses, with larger frames. The child is never seen now to squint. Vision R. E. $\frac{6}{6}$, L. E. (the originally fixing eye) $\frac{6}{9}$ easily, $\frac{6}{6}$ partly.

October 24, 1902.—At my request, the mother brings the child (aged now 9 years 10 months) to me again for examination. She has grade III. binocular vision, with a good amplitude of fusion. She never squints now, either with or without her glasses. She must always wear glasses, of course, on account of her refractive error, but I have given her permission to dispense with them during her dancing lessons and gymnasium practice.

Case A, 437. *January* 18, 1900.—I saw a boy, aged 3 years 7 months, at the West Ham Hospital. He began to squint, aged 2 years 10 months, during convalescence from scarlet fever. Squint was at first occasional. It became constant after a few weeks. C. S. R. E. 42°. Good fixation R. E. Ivory ball test shows that R. E. has at least $\frac{6}{12}$ vision. Ordered, atropine for retinoscopy.

January 25.—C. S. R. E. 44° with atropine. Retinoscopy—R. E. + 4·75 D. sph. + 1·75 D. cyl. ax. 70° down and in. L. E. + 4·75 D. sph. + 1 D. cyl. ax. 70° down and in. Ordered, spectacles 0·75 D. less than the retinoscopy: also, guttæ atropinæ L. E. *only* every morning.

February 8.—The boy uses the unatropised R. E. always, both in near and distant vision. C. S. 32° with glasses. Ordered, stop drops.

During March and April exercises with the amblyo-

scope were carried out six times. They resulted in a fairly well-developed fusion sense with an amplitude of fusion of about 10°.

April 26.—C. S. alternating 27°.

June 7.—C. S. 21°. Fusion training.

(In a case in which I have succeeded in developing the fusion sense, but in which there is still a considerable degree of deviation, the next step in the treatment will depend upon measurements of the angle of the deviation at each visit. If this is decreasing at a reasonable rate, the patient is given fusion training once every four or five weeks. This is quite sufficient to preserve the fusion faculty, while optical treatment is being tried. If, however, the improvement ceases, so that nothing more is to be expected from the wearing of glasses, operation becomes necessary.)

July 12.—C. S. 16°. Fusion training.

August 16.—C. S. 12°. Fusion training.

September 27. No squint with glasses. Good binocular vision. The mother says that the eyes became straight soon after the last visit. He squints, sometimes when the glasses are off. When the glasses are taken off here, in the light he does not squint; but, in the dark room, he turns in one or other eye most of the time.

July 3, 1902.—The boy lost his glasses yesterday. There is no squint in distant vision, even without the glasses. But when he is told to pick out the letters on a card he uses his L. E. and turns in the R. E. to an extreme degree. He then covers the R. E. with his hand. Ordered, repeat glasses.

CASE A, 503. *June* 7, 1900.—A girl,[1] aged 5 months,

[1] On October 23rd, 1902, a sister of this patient, an infant aged 14 weeks, was brought to me at the West Ham Hospital. She had C.S.L.E. about 30°. On October 30th retinoscopy under atropine + 6 D. sph. each eye. I

brought to me at the West Ham Hospital. She has squinted constantly with the L. E. since she was twelve weeks old. She has C. S. L. E. about 25°, variable. Fixation lost L. E. Abversion L. E. full. Ordered, continuous occlusion R. E., by a pad and bandage, for fourteen days : also. ung. atropinæ 1 per cent. thrice daily, for both eyes, for retinoscopy.

June 21.—Steady central fixation L. E. Under atropine, angle of squint varies from about 20° to 40°. Retinoscopy R. E. + 5·5 D. sph., L. E. + 6·5 D. sph.

Ordered, spectacles, of the pattern described on page 102. R. E. + 5 D., L. E. + 6 D. Ordered also, ung. atropinæ 1 per cent., to be put into the R. E. *only* every morning.

August 16.—Glasses worn well, child seems to be quite unconscious of their presence. There is no squint in distant vision. When looking at anything near at hand the infant turns in the R. E. (atropised). Ordered, stop the atropine.

October 8.—No deviation with glasses. When glasses are taken off L. E. turns in to a variable degree—about 30° usually.

May 9, 1901.—No Squint with glasses.

October 3.—Glasses too small. Ordered, repeat them.

August 28, 1902.—Glasses were lost ten days ago. The child is not squinting now, even without the glasses. Ordered, repeat the glasses.

September 25.—No squint with glasses or without

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ordered + 5·5 D. and sent her to Mr. Hawes, of Leadenhall Street, who fitted spectacles of the pattern described on page 105. When I saw her on November 6th she had had the spectacles two days. She was wearing them quite happily. Atropine was used for the fixing eye only until the squint alternated. I last saw her on June 7th, 1904, she had no deviation while wearing the glasses, and prism test in the dark room showed that the natural development of binocular vision was proceeding.

them. When a lighted match was held before the child's eyes in the dark room, she of course looked at it. A prism, apex towards the nose, was then slipped before one eye. This eye was immediately seen to make a slight inward rotation (in order to blend the images of the light), showing that the child had binocular vision.

(I do not think that hypermetropes, during the first three or four months of life, as a rule make any prolonged effort of accommodation in the interests of sharp vision. But some infants, this girl, for example, evidently do so. The abnormal accommodative effort caused an abnormal dynamic convergence, which was soon succeeded by a static convergence, before the period at which the fusion sense, normally, has made much progress in development. By giving her a pair of spectacles, and so relieving the strain on the accommodation, the visual axes were, in the course of four or five weeks, brought back to parallelism. This allowed the natural development of the fusion sense to take place. The child, in spite of the high refractive error, is perfectly cured, whether she wears glasses or not. Nothing but an actual muscular paralysis will ever make this child squint again.

The case of this child's sister is exactly similar.

If I had followed the practice recommended in the text-books, of leaving the case until the child was "old enough to wear glasses," she would then have had an incurable squint, and almost total blindness of the left eye.

Case B, 227.—*May* 16, 1900.—A girl, aged 16 years. Her right eye is nearly blind. It turns out and down, and is very prominent—a hideous deformity—the result of two tenotomies six years ago.

History.—She had whooping-cough soon after she was one year old. During convalescence the R. E. turned in towards the nose. She was taken to an

ophthalmic surgeon at once. Being considered to be too young for glasses, she was ordered atropine drops for both eyes. The drops were used for *both* eyes, for from a year and a half to two years. (How often one hears this disastrous tale!) Soon after she was three years old she was ordered spectacles. At the age of 10 years, the right eye was operated upon, in London (tenotomy of internal rectus). A year later this eye was again operated upon in Germany. After the second operation the R. E. was "straight." Soon after that it began to turn out and down. This deformity has gradually got worse.

At present R. E. is very prominent: it turns out 28° and down 10°. With this eye she can just distinguish hand movements close to the face. Power of adversion R. E. is absent. She is wearing spectacles + 1·75 D. sph. each eye. Vision L. E. $\frac{6}{6}$ both with and without glasses. Retinoscopy—with homatropine L. E. + 2·25 D., R. E. + 3·5 D. at approximate macula.

Of course, the blindness of the R. E. is now quite incurable. With a view to remedying the deformity, I advised that the divergence should be dealt with by operation first, and the vertical deviation at a subsequent operation.

May 21.—Having cocainised the eye, I stripped up from the globe the membranes on the nasal side, from near the edge of the cornea to the sunken caruncle. I found the internal rectus muscle behind the sunken caruncle, near the inner wall of the orbit. It was not attached to the globe in any way. As the muscle was much wasted, I decided to advance the conjunctiva, and capsule of Tenon also. I seized all these structures with forceps, and drew them between the jaws of a Prince's advancement forceps. I secured these structures, by my usual method, to the circum-corneal fibrous tissue, bringing the eye into a position of very slight convergence.

June 16.—I advanced the right superior rectus muscle.

June 30.—There is still some redness of the eye. The advancements have drawn back the eye into its proper position in the orbit. In ordinary directions of the gaze the eyes appear quite straight. But, when the patient looks more than about 15° to the right, the right eye ceases to follow the movement of the left. This is because, owing to the wasted condition of the internal rectus muscle, I did not dare to rely upon the muscle alone, but was compelled to advance the membranes also. But it is easy for the patient to conceal this defect, by avoiding wide excursions of the eyes. I ordered pince-nez + 2 D. sph.

July 5, 1902.—Patient does not wear her glasses out of doors. She always wears them indoors. The eyes are quite natural in appearance.

CASE D, 81. *September* 27, 1899.—Boy, aged 6 years 5 months, seen at Moorfields. C. S. L. E. 27°. Abversion D. E. very slightly deficient. He began to squint during convalescence from measles, aged $2\frac{1}{2}$ years. He has had no treatment. Ordered, atropine for retinoscopy.

September 30.—Retinoscopy each eye + 3 D. sph. + 0·75 D. cyl. ax. vert. Vision R. E. $\frac{6}{6}$, L. E. $\frac{6}{36}$. Ordered, spectacles 0·5 D. less than the retinoscopy: also, guttæ atropinæ 1 per cent. every morning R. E. *only*.

November 4.—Boy uses R. E. (atropised) in distant vision and L. E. (unatropised) in near vision. C. S. L. E. 16°. V. L. E. $\frac{6}{18}$. Ordered, continue drops R. E. only.

January 3, 1900.—C. S. L. E. 11°. V. L. E. $\frac{6}{12}$ partly. Boy too old for fusion training. Ordered, continue for four months.

May 2.—C. S. L. E. 6°. V. L. E. $\frac{6}{12}$ partly. Ordered, continue drops for R. E. for two months more.

July 4.—C. S. L. E. 5°. V. L. E. $\frac{6}{12}$ partly. Ordered, stop drops.

February 6, 1901.—With the glasses there is no apparent squint, but the mirror test reveals a convergent squint of 2°. C. S. about 15° when the glasses are taken off. V. R. E. $\frac{6}{6}$., L. E. $\frac{6}{12}$ partly.

CASE D, 734. *November* 29, 1901.—Girl, aged 4 years 8 months. C. S. R. E. 22°. Abversion perfect. The power of central fixation of R. E. is lost. The eye is so blind that, when the good eye is tied up, she is unable to see a penny on the floor at her feet. She hears it drop, and goes down on her knees to feel for it. The mother says that the squint began at about 2 years of age, during an attack of scarlet fever. She says she repeatedly spoke to the doctor about it, but he told her to "wait to see if the child would grow out of the squint!" Ordered, atropine for retinoscopy.

December 3.—C. S. R. E. 26° with atropine. Retinoscopy R. E. + 3 D. sph. at approximate macula. L. E. + 3 D. sph. Ordered, spectacles + 2·5 D. sph. each eye: also L. E. to be continuously occluded by a gauze pad and strapping.

January 3, 1902.—When the eye was at first tied up, she used to fall over things, the mother says. Later, she could see to run about very well. Child has now central fixation R. E. Ordered, discontinue pad; guttæ atropinæ 1 per cent. to be put into L. E. *only*, every morning. Child to go to school and use the eyes in near vision as much as possible.

February 6.—Child uses (atropised) L. E. in distant vision, and (unatropised) R. E. in near vision. Ordered, continue atropine L. E. *only*.

May 2.—With glasses child uses (atropised) L. E. in distant vision, and (unatropised) R. E. in near vision. When the glasses are off she uses the R. E. and turns in L. E. always. C. S. R. E. 16° with glasses. Ordered, continue atropine L. E.

October 3.—Vision R. E. with glasses $\frac{6}{12}$ easily. C. S. R. E. 13°. Ordered, continue atropine L. E. only.

December 2.—Child uses (unatropised) R. E. now always, both in near and distant vision, and turns in the (atropised) L. E. C. S. L. E. 14°. Vision R. E. $\frac{6}{6}$ with difficulty ; $\frac{6}{9}$ easily ; L. E. $\frac{6}{6}$.

(In private practice, I should have aimed at a perfect cure of the squint, by training the fusion sense. But, in a crowded hospital clinic, it is, unfortunately, not possible to find time for fusion training, except in a very small proportion of the cases. But it is something to have restored the sight of the blind eye.)

CASE A, 541. *August* 9, 1900.—A boy, aged 16 years, came to me at West Ham Hospital. He had squinted with L. E. since infancy. He had worn glasses since he was 4 years old. He was wearing + 3·5 D. sph. each eye. Vision with glasses, R. E. $\frac{6}{6}$, L. E. $\frac{6}{60}$. He had C. S. L. E. 11°, with his glasses. His glasses were found to be suitable. Adversion L. E. good. Dynamic convergence good. It was, of course, much too late to attempt to restore the sight of the left eye. He wished to have the deformity removed.

Under cocaine and supra-renal extract, I performed complete central tenotomy of left internal rectus by the method described on page 216.

August 16.—Scarcely any redness remaining. C. S. L. E. 3°, with his glasses. This small squint is quite masked by the angle gamma.

May 8, 1902.—Patient has, with his glasses, C. S. L. E. 4°. There is no noticeable deformity.

CASE B, 19. *November* 12, 1895.—A girl, aged 3 years 1 month. She began to squint during convalescence from measles, aged 1 year 10 months. Squint was, at first, occasional. It soon became constant. She has now C. S. alternating 36°. Ordered, atropine for retinoscopy.

November 16.—C. S. alternating 32°, with atropine. Retinoscopy, each eye, + 5 D. sph. + 1 D. cyl. ax. vert. Ordered, glasses + 4·5 D. sph. + 1 D. cyl. ax. vert.

November 27.—C. S. alternating 28°, with glasses. Fusion training with amblyoscope.

December 5.—Fusion training. Child easily fuses images.

December 18.—Fusion training.

January 10, 1896.—Fusion training. Child has an amplitude of fusion of at least 10°. C. S. 18° with glasses.

January 18.—Fusion training.

February 12.—Fusion training. Child can follow the images through a range of 15° or 20°. C. S. 12°.

March 11.—Fusion training. C. S. 10°.

March 30.—No squint with glasses.

October 8.—No squint. Glasses worn comfortably.

August 21, 1897.—Child never squints now. Ordered, similar glasses in larger frames.

July 7, 1899. The girl never squints now, even when the glasses are taken off; but she must, of course, continue to wear the glasses on account of her high refractive error.

CASE B, II., 57. *January* 13, 1902. A girl, aged 11 years 5 months. *History.* She began to squint with L. E. when she was 6 years old. She has squinted with this eye constantly ever since. When she was about 8 years old she had glasses. In April, 1899, the left internal rectus muscle was tenotomised, and in June, 1899, the right also.

She now has convergent squint L. E. 15°, with the glasses. Her vision, with her glasses, is $\frac{6}{6}$ each eye. She has a faint homonymous diplopia when she looks for it. On examining her fusion sense with the amblyoscope she readily blends images, but has almost no amplitude of fusion.

January 24.—Retinoscopy under atropine, each eye, + 3·5 D. sph. + 1·5 D. cyl. ax. 30° down and out. This very nearly corresponds with the glasses she has been wearing.

February 12.—By my direction, the glasses have been left off for the last twenty-four hours. Without glasses, R. E. fixing, the deviation is 22°. L. E. fixing, the deviation is 35°. (Approximate estimation by mirror test).

I advanced the left external rectus muscle (under cocaine), of course without tenotomising the internal rectus. Eyes "straight" after operation.

February 19.—Removed sutures. Mirror test shows binocular fixation.

February 28.—The patient had binocular vision, both with and without her glasses, but the amplitude of fusion is very small.

June 18.—Since the operation I have, by way of experiment, attempted to increase the amplitude of fusion, but of course without success. She has now just the same small degree of fusion sense which she had when she began to squint, and which she has had ever since—neither more nor less. But an accurately performed advancement has enabled her to use this feeble fusion sense. The eyes are exactly straight and the child has binocular vision.

The reason of the good vision in the deviating eye is the unusually late onset of the deviation. Amblyopia from disuse is scarcely ever acquired after six years of age.

CASE B, 33. *May* 5, 1896.—Boy, aged 2 years 7 months. The right eye turned in suddenly a little before he was two years old. He has squinted constantly ever since. C. S. R. E. 27°. Central fixation R. E. With R. E. he can with difficulty see the 1½ inch ivory ball at four yards. Abversion each eye perfect. Ordered, atropine for retinoscopy.

May 12.—Under atropine C. S. R. E. 30°. Retinoscopy, each eye + 2·25 D. sph. Ordered, spectacles + 2 D. sph. constant wear: also guttæ atropinæ 1 per cent. L. E. *only*, every morning.

July 3.—Child uses the R. E. (unatropised) in near vision and the L. E. (atropised) in distant vision. C. S. R. E. 21° with glasses. Ordered, continue.

August 26.—Same. C. S. R. E. 18°. Ordered, continue.

October 12.—The boy uses the (unatropised) R. E., and turns in the (atropised) L. E., nearly always now, even in distant vision. Ordered, continue.

November 6.—C. S. L. E. always now 12°. Ordered, stop atropine.

November 27.—Squint alternates now 9°. Fusion training with amblyoscope.

December 4.—Fusion training. Child readily blends images, but he has at present no amplitude of fusion.

December 10.—Fusion training.

December 14.—Fusion training.

December 16.—Fusion training.

December 19.—Fusion training. Good amplitude of fusion.

January 22, 1897.—No squint.

August 12.—Child wears his glasses. No squint.

October 25, 1898.—Child never squints now.

July 18, 1899—The boy does not squint even when the glasses are taken off.

June 13, 1902.—Ordered, atropine for retinoscopy.

June 18.—Retinoscopy R. E. + 1·75 D. sph. + 0·25 D. cyl., L. E. + 1·75 D. sph. Vision $\frac{6}{6}$ each eye. Ordered, glasses for near work + 1·75 D. sph. No glasses to be worn out of school. To be seen again in three months.

September 22.—The boy gets on well without glasses. He prefers not to use them even for school work. He never squints now.

CASE B., 165. *July* 19, 1899.—Girl, aged 3 years 5 months. Since earliest infancy R. E. had turned out occasionally: worse during the last year. Never any diplopia. Always a delicate and timid child.

The eyes sometimes fix binocularly, but more often R. E. is widely divergent. When the child is spoken to the eyes recover their normal relative directions immediately, but when she looks at a near object the R. E. is usually allowed to diverge. Dynamic convergence very deficient. All separate movements of each eye perfect. With L. E. the child easily sees the ½ inch ivory ball at six yards. With R. E. she can with difficulty see the 1½ inch ball at three yards. R. E. has not lost central fixation. Amblyoscope test shows that the child has some slight degree of fusion sense.

July 26.—Retinoscopy under atropine, each eye + 1·25 D. sph. No glasses ordered. Ordered, guttæ atropinæ sulph. 1 per cent. L. E. *only* every morning.

August 22.—Child now uses L. E. (atropised) for distance, and R. E. (unatropised) in near vision. Ordered, continue.

October 27.—Child uses R. E. in near vision and sometimes in distant vision also. With R. E. she can easily see the ¾ inch ivory ball at six yards. Ordered, atropine L. E. only every morning, first seven days in each month, for six months.

September 20, 1900.—Divergence is nearly alternating. Still some preference for fixing with L. E.

July 16, 1902.—Divergence almost always present now. V. R. E. $\frac{6}{6}$ partly, V. L. E. $\frac{6}{6}$.

July 29.—Advancement R. internal rectus muscle, under cocaine. Patient kept in bed with both eyes bandaged for one week after. Stitches then removed. Two days later all dressings discontinued.

August 26.—Eyes "straight." Child blends images except on looking to extreme R.

(The feeble degree of fusion sense which developed

in early childhood was not sufficient to prevent the deviation, or even to cause diplopia, but now that the deviation has been overcome by operation it enables her to fuse the two images.)

CASE B, 166. *July* 19, 1899.—Girl, aged 8 years 2 months, sister of the preceding case. Alternating divergent squint of variable degree, usually about 35°. Association between accommodation and convergence almost absent. Separate movements each eye normal, except adversion, which is slightly deficient. Fusion sense absent. H.m. 0·5 D. No As. V. each eye $\frac{6}{6}$. Child has worn spectacles + 0·75 D. sph. for about three years. Recommended advancement of one or both internal recti for cosmetic reasons. No glasses required.

August 22.—Advancement R. internal rectus, under cocaine (of course without tenotomy of externus.)

November 8.—Advancement L. internal rectus.

December 20.—No noticeable deformity now. Eyes appear to be quite normally directed. Mirror test, however, shows that there is sometimes slight divergence and sometimes slight convergence. Of course there is no fusion: she really uses the eyes alternately, although she appears to use them together.

October 5, 1904.—Condition as at last visit five years ago.

CASE A, 79. *December* 5, 1895. Girl, aged 16 years. Eyes are already under atropine. L. E. is widely divergent. When told to look at a near object she can, by an effort, overcome the deviation, but the eye soon diverges again. Adversion slightly deficient. Patient is known to have been "short-sighted for some years; getting worse." Four or five years ago L. E. turned out, occasionally at first, but during the last year constantly. She often sees double. Small myopic crescent each eye. Choroidal vessels seen. Retino-

scopy R. E. — 6·5 D. sph. V. $\frac{6}{6}$ partly. L. E. — 6·5 D. sph. — 1·75 D. cyl. ax. 15° down and out. V. $\frac{6}{9}$. Ordered, full correction to be worn constantly.

February 13, 1896.—When she first began to wear the glasses they made her head ache, but she soon became accustomed to them. Now she finds them quite comfortable. No divergence now. When she is tired she "squints and sees double for a moment until the eyes come straight again." While wearing the glasses either eye diverges when screened, about 20°.

October 8.—Glasses very comfortable. No headaches. No divergence. Behind Maddox rod either eye turns out 12°, varies slightly.

September 23, 1897.—Retinoscopy under atropine shows that the myopia has increased 0·75 D. Ordered, continue same glasses.

July 20, 1899. She is never seen to squint, but says that she occasionally sees double for a moment. Behind Maddox rod either eye diverges 8° or 9°.

July 27. Retinoscopy under atropine. R. E. — 7·5 D. sph. V. $\frac{6}{6}$ L. E. — 7·5 D. sph. — 1·75 D. cyl. ax. 12° down and out. V. $\frac{6}{9}$. Ordered, full correction, to be worn constantly.

August 20, 1903.—Eyes comfortable. Never any divergence. Exophoria is now only 2°.

August 27.—Retinoscopy under atropine shows that the myopia has increased less than 0·5 D. during the last four years.

CHAPTER XI

HETEROPHORIA.

If a person, with a perfectly normal pair of eyes, looks steadily at any object, both visual axes will continue to be accurately directed to that object, even though one eye be shaded. In other words, his perfectly balanced motor coördinations are able to maintain the normal relative directions of the eyes, even when the controlling influence of the fusion sense is temporarily withdrawn. This state of perfect oculo-motor equilibrium is called *orthophoria.*

Heterophoria is the name given to the condition of imperfect oculo-motor balance. There is here a *tendency* for the eyes to deviate from their normal relative directions. Ordinarily, however, this tendency is kept in check by the fusion sense, so that there is no squint. But if binocular vision be temporarily rendered impossible—*e.g.*, by covering one eye—this tendency gives rise to an actual deviation.

Heterophoria of sufficient degree to cause trouble is not very common. Of those who suffer from "asthenopic symptoms," in only a very small proportion of cases are the symptoms found

to be due to heterophoria. Occasionally, however, one meets with a patient who complains of pain and discomfort in the eyes, and whose refraction has been repeatedly examined, and who has for years worn glasses to correct some unimportant refractive error, without any relief to his suffering. Such a patient usually has a heterophoria, the correction of which immediately and permanently removes his trouble.

Heterophoria may perhaps be due to a muscle or group of muscles being too weak or too strong for the opponents, or to an abnormal position of insertion of a tendon, whereby the muscle acts at less or more than its normal mechanical advantage, or to a muscle or group of muscles being too feebly, or too powerfully, innervated. As a rule, we are unable to determine whether the fault lies in the muscles themselves, or in their innervations. We can only say that certain *actions* are deficient or excessive. Heterophoria is essentially a motor anomaly.

Squint, on the other hand, is essentially due to a defect of the fusion faculty. In the presence of this fundamental cause, heterophoria may give rise to a permanent squint with suppression of one image: not otherwise.

There is an apparent exception to this rule—a person who has previously enjoyed perfect binocular vision may have the visual acuity of one eye so lowered by progressive myopia, injury, or

disease as to render binocular vision impossible. Any heterophoria which may be present will then cause a manifest squint, although the cerebral faculty of fusion remains perfect.

Heterophoria is the generic name, invented by Stevens, for all latent tendencies to deviation. Distinctive names are employed to indicate the direction of the tendency :—

Esophoria is a tendency to abnormal static convergence of the visual axes.

Exophoria is a tendency to divergence of the visual axes.

Hyperphoria is a tendency of the two eyes to rotate vertically in opposite directions, so that one visual axis shall lie in a higher plane than the other. The eye which tends to turn upwards is called the hyperphoric eye.

Cyclophoria is a tendency to abnormal rotation of one or both eyes round a fore-and-aft axis, so that what should be the vertical meridian of the eye shall be no longer parallel to the median plane of the head. A tendency for the vertical meridian of the eye to lean away from the median plane is called plus cyclophoria. A tendency in the opposite direction is called minus cyclophoria.

Pseudo-heterophoria.—In a case of uncorrected ametropia there is frequently an apparent heterophoria which disappears when the appropriate correcting glasses are worn. The term heterophoria should be reserved for cases in which the

anomaly persists after optical correction of any refractive error which may be present.

If the patient be ametropic, he should wear an exact correction of his ametropia during the examination. But this spurious heterophoria does not always disappear immediately on correcting the refractive error. So that, if the latter be considerable, one should not immediately conclude that any heterophoria which may be found is genuine. The results should be checked by a second examination after glasses correcting the refractive error have been worn for several weeks.

The *symptoms* of heterophoria are those of "eye-strain" in general—frontal headache coming on towards the end of the day; pain in the eyes after watching anything intently, *e.g.*, a play; migraine; dizziness (especially associated with hyperphoria); conjunctival hyperæmia, &c. In the higher degrees of heterophoria momentary deviation with diplopia is not uncommon.

Asthenopic symptoms, which do not yield to accurate optical correction of any refractive error which may be present, should always lead to investigation of the motor balance of the eyes, if this has not already taken place.

People vary greatly in their susceptibility to suffering as the result of heterophoria, just as they do in the case of refractive error. Other things being equal, hyperphoria is the form of heterophoria which is most likely to cause trouble,

and esophoria the least. It is not uncommon to see a patient who has several degrees of esophoria, and who is quite unconscious of any defect; whereas few men can support a hyperphoria of more than one degree without inconvenience.

The importance of a case of heterophoria is proportionate to the trouble which it causes. A case which gives rise to no symptoms requires no treatment.

Heterotropia.—A person whose fusion sense has developed perfectly, but who has a very high degree of heterophoria, will be able (with more or less suffering) to keep this deviation-tendency in check during the adaptable and vigorous period of childhood and youth, but, when he exchanges school life for some more trying and less healthy occupation, he may find himself unable to continue the struggle, in which event his heterophoria gives rise to an actual deviation. He then loses his asthenopic symptoms, but he suffers from diplopia, which is usually so annoying that he is glad to shade or close one eye. The degree of the manifest deviation increases during the first few weeks or months, after which it becomes stationary. The term *heterotropia* should be reserved for this rather rare condition, as it is obviously a further stage of heterophoria, and not a true squint nor a paralysis. The case of Mr. S. H., page 195, is a typical example.

The methods of testing the muscular balance

of the eyes.—In a case of heterophoria, under ordinary circumstances, the desire for binocular vision prevents the eyes from deviating from their normal relative directions. But if, by artificial means, the image formed in one eye be so altered in appearance or position as to make fusion with the other unaltered image impossible, the control of the fusion-sense is suspended. The heterophoria then gives rise to a manifest deviation. The altered image in the

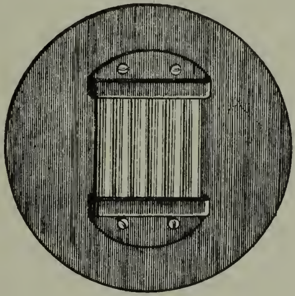

FIG. 19.

deviating eye is not suppressed as in a case of squint. The diplopia, therefore, gives an easy means of ascertaining the direction and degree of the deviation. This is the principle on which all subjective tests for heterophoria are based.

The *instruments* required for the tests which I am about to describe are the Maddox rod and tangent scale, the Maddox double prism, an adjustable trial frame, test cards, and a set of prisms whose axes are accurately marked. A rotary prism also is very useful.

The Maddox rod (fig. 19). A transparent round glass rod is, in effect, a very strong cylindrical lens. Rays of light, therefore, which pass through it are dispersed in one plane only, at right angles to the axis of the rod. So that if a point of light be looked at through this rod, it will appear as a long narrow band of light. For the sake of convenience, half a dozen of these pieces of glass rod are fixed, side by side, in a metal disc of such a size as to fit into an ordinary trial frame. The rods are generally made of red glass, to increase the contrast between this band of light and the true image. An equally good plan is to have the rods made of colourless glass and to put a plane red glass before the other eye.

In looking at a flame, with the rod before one eye and the other eye naked, the naked eye will of course see the flame and the surrounding objects; but, to the rod-clad eye, the flame will appear as a long streak of light, and less luminous objects will not be visible at all. It is not possible to blend two such dissimilar images as the flame and the streak,[1] so the function of fusion is temporarily

[1] The images should be differently coloured. The rods should be carefully fitted side by side, so that it is not possible to see between them. The source of light at the zero of the scale should, if possible, be a frosted incandescent electric lamp. Failing this, any bright flame, enclosed in a tin chimney having a hole of about $1\frac{1}{4}$ inch diameter in one side of it, will serve the purpose. The room should not be too brightly illuminated. As the rods

suspended, and the eyes are merely controlled by their motor coördinations. If there be no motor anomaly, the streak, seen by the rod-clad eye, will appear to pass through the flame, seen by the

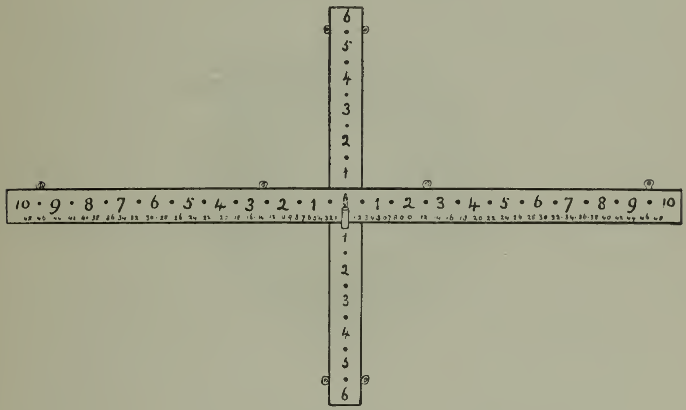

FIG. 20

naked eye. But if there be any heterophoria it will now be able to cause the eyes to deviate, the relative positions of the streak and light indicating the direction and degree of the deviation.

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produce extreme distortion in one direction only, a well marked vertical line can be seen through the rods when their axes are horizontal, and a horizontal line can be seen when their axes are vertical. Care should therefore be taken that there be no prominent horizontal or vertical lines near the centre of the field of vision. For this reason, the paper on which the tangent scale is printed should be of nearly the same colour as the background on which it is hung: or the large figures may be marked on the wall itself. With these precautions, I have always found the rod test quite reliable.

The *tangent scale* is shown in fig. 20. The large figures on the horizontal and vertical scales denote tangents to degrees at a distance of 5 metres. A small electric or other light is placed at the zero of the scale.

FIG. 21.

The Maddox *double prism* (fig. 21) consists of two prisms, each of 4°,[1] cemented base to base. When this double prism is placed with its apices vertical, before one eye, so that the line of junction of the bases crosses the pupil horizontally,

[1] To avoid confusion, the *strength of a prism* is always, in this book, denoted by the *number of degrees which it deflects a ray of light.* Chromatic dispersion produces no appreciable error in the weak prisms used in ophthalmology.

An optician usually numbers a prism according to the width of its geometrical angle (the angle between the two plane surfaces). The refracting power of such a prism varies according to the kind of glass of which it is made. For practical purposes it may be taken as half the geometrical angle. For instance, a prism which deflects a ray of light to the extent of 4° will have a geometrical angle of about 8°.

two false images of any small object will be seen, one above and the other below its true position. If now the other (naked) eye be opened, it will see the real image of the object mid-way between the two false images. The false images are not changed in appearance, but the vertical displacement of each is too great to admit of the true image being blended with either of them. The eyes are thus temporarily deprived of the control of the fusion sense and abandoned to their motor coördinations, just as in the last test.

In looking at a horizontal line, the true image can of course not be made to approach either of the two false images ; but care must be taken that there be no long vertical line near the centre of the field of vision.

The *test cards* which I use consist of two pieces of strong white cardboard each 2 feet square. Number I. card has in its centre a straight black line 2 inches long. In the centre of Number II. card are ten letters of "Pearl" type having a large capital O in the middle. The cards are meant for use at the reading distance. The reason for making them so large is that the objects shall be seen in the centre of a blank field with no edges near to solicit fusion.

In the *examination* I begin with the Maddox rod. The patient is seated before the tangent scale, at a distance of 5 metres from it. If he be not absolutely emmetropic, he wears correcting

Number I. test card.

FIG. 22.

sreox **O** aveon

Number II. test card.

FIG. 23.

lenses in the trial frame in every test. The frame is adjusted so that the lenses are accurately centred for distant vision. The rod, with its axis horizontal, is put in the frame before the right eye. The light, at the zero of the tangent scale, is switched on. If the vertical streak, seen by the right eye, appears to go through the light, seen by the left eye, the patient has no esophoria or exophoria in distant vision. Now rotate the rod so that its

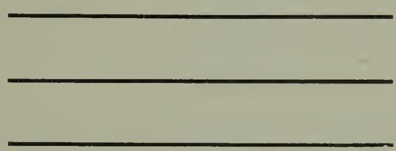

FIG. 24.[1]

axis is vertical. If the horizontal streak, now seen by the right eye, appears to pass through the light seen by the left eye, the patient has no hyperphoria in distant vision.

Now remove the rod and replace it with the double prism, with apices vertical (line of junction of bases horizontal). Adjust the trial frame for near vision. Let the patient hold in his hand Number I. test card, with the line horizontal.

[1] This and the two succeeding figures are taken from a paper on "Insufficiency of the Obliques," by Dr. Savage, of Nashville, U.S.A., "Archives of Ophthalmology," January, 1891. "Ophthalmic Myology," by the same author, contains the fullest account of cyclophoria which has hitherto been published.

He will see two false images of the horizontal line with the prism-clad eye, and between them, he will see the true image with the naked eye. If the middle line appears equidistant from each of the other lines, and has its ends level with their ends (fig. 24) there is no hyperphoria, esophoria, or exophoria in near vision. If the middle line appears parallel to the other two lines, as in this figure, there is no cyclophoria. The patient's oculo-motor equilibrium, therefore, is perfect in every respect.

If, however, any anomaly be found during these proceedings, further examinations will be required.

In the *distant vision test* with the Maddox rod, axis horizontal, before the right eye, if the vertical streak lies to the right of the light (homonymous diplopia), there is esophoria. If it lies to the left of the light (crossed diplopia), there is exophoria. The figure on the tangent scale, through which the vertical streak passes, numerates the degree of the defect. As a control test, change the rod from the right eye to the left. The position of the streak also changes over, that is, it still shows the same kind of diplopia. The degree of heterophoria indicated is the same as before. Now place before one eye a prism of the same degree as the defect, base out in esophoria, base in in exophoria. This should cause the streak to pass through the light.

In using the rod, axis vertical, before the right

eye, if the horizontal streak is seen below the light, this indicates that the right eye tends to turn upwards relatively to the left eye (right hyperphoria). If the streak is seen above the light, the left eye tends to turn upwards relatively to the right eye (left hyperphoria). The figure, on the vertical scale, which the streak appears to cross, numerates the degree of the hyperphoria. Now change the rod from the right to the left eye. Almost invariably, if the right eye saw the streak above the light, the left eye will now see it below, and *vice versâ*. That is to say, the hyperphoria is comitant. The result should be checked by neutralising the hyperphoria with a prism of the strength indicated by the position of the streak.

In rather rare instances it happens that each eye, in turn, rotates upwards behind the rod (double hyperphoria).

In the *near vision test* with the double prism before the right eye, and the Number I. test card, the middle line, seen by the left eye, should be equidistant between the two false images, seen by the right eye. If it lies nearer the upper false image, there is right hyperphoria. If it lies nearer the lower false image, there is left hyperphoria. The prism, base down, before the hyperphoric eye, which places the line half-way between the two false images, will serve to measure the degree of the anomaly.

If the three lines are not all level with each other at the ends, hand the patient Number II. test card and tell him to read the letters on it. The small letters are to ensure a normal effort of accommodation. If presbyopic, the patient is allowed glasses. He will see the true image of the object between its two false images. In orthophoria the three images will be in the same vertical line. If the middle image, seen by the left eye, is to the left of the two false images, the

FIG. 25.

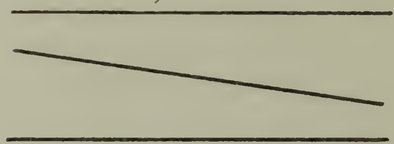

FIG. 26.

patient has esophoria in near vision. If it is to the right, he has exophoria in near vision. The prism, axis horizontal, which will bring the three O's in line, is a measure of the defect. In near vision it is better to measure with prisms than to use any kind of small tangent scale, because with the former the distance from the eye is of no consequence, whereas with the latter the smallest

variation introduces an error. The study of esophoria and exophoria in near vision is intimately associated with that of convergence anomalies.

When the patient looks at the horizontal line on Number I. card, with both eyes open and the double prism before the right eye, the three lines should be parallel (fig. 24). If not, there is cyclophoria. Suppose the middle line, seen by the left eye, seems to dip down to the left as in fig. 25, this shows that the vertical meridians of the eyes are leaning in the opposite direction, towards each other (minus cyclophoria). If the middle line, seen by the left eye, appears to dip down to the right as in fig 26, there is plus cyclophoria.

Prism duction should always be investigated in any case in which heterophoria has been found.

Seat the patient at a distance of five or six metres from a candle flame. Let him wear a trial frame. While he looks steadily at the light, put a 1° prism, apex up, before the right eye. Gradually increase the strength of the prism, until the highest prism is found which the patient can bear without seeing double. This indicates the extreme range of superduction of the right eye. Now test the superduction of the left eye. The subduction of each eye is similarly tested, with prisms apex down. The power of binocular abduction is tested with prisms apex out. Binocular adduction is so intimately associated with accommodation that an attempt to measure it with prisms (which

cause the eyes to converge without accommodating) gives very variable and misleading results.

The normal limits of prism duction are as follows:—

Superduction ...	...	$1\frac{1}{2}°$ to $2\frac{1}{2}°$
Subduction ...	...	$1\frac{1}{2}°$ to $2\frac{1}{2}°$
Abduction ...	...	$4°$ to $5°$

No amount of practice appears to increase the duction power in these three directions. Convergence, on the other hand, can nearly always

FIG. 27.

be much increased by practice. As the degree of prism-duction does not vary from time to time, and is independent of voluntary effort on the part of the patient, the information obtained is reliable.

A rotary prism (fig. 27) is very convenient for measuring duction. It consists of two prisms of equal strength, mounted in a metal disc in such a position that the apex of each coincides with the base of the other. In this position they, of course, neutralise each other. By a mechanical arrange-

ment, the two prisms can be rotated in opposite directions at equal rates. The strength of the compound prism can be thus gradually increased from zero up to the combined strength of the two components.

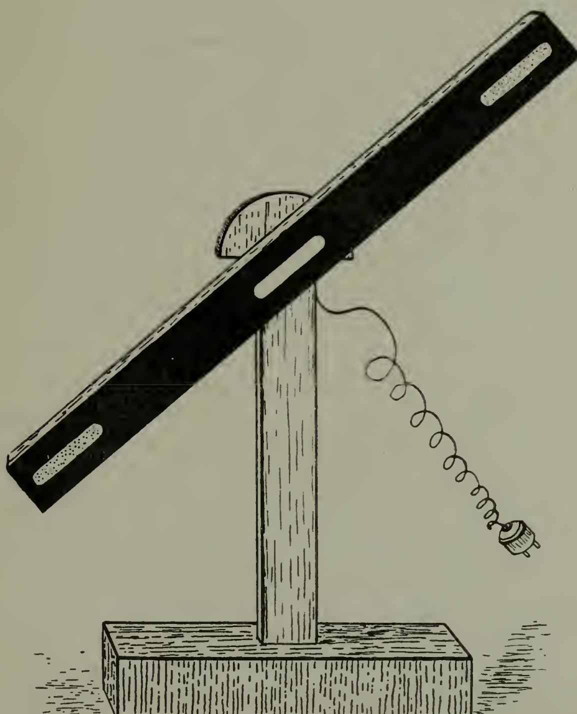

FIG. 28.

Phorometer.—Fig. 28 shows an instrument which I have found very useful for measuring heterophoria. A rod-shaped wooden box 24 inches by 2 inches by 2 inches, is supported on a stand by a horizontal bolt, so that it is free to rotate in a vertical plane. (A two-foot length

of brass optical tube would do as well as the rod-shaped box). In the face of this rod-shaped box are three openings, each 3 inches by $\frac{5}{8}$ inch. In the centre opening is fitted a piece of frosted red glass.[1] In each of the two other openings is a piece of frosted green glass. In the box, behind each glass, is a small electric lamp having a tin reflector behind it to increase the illumination. The box is ventilated by holes in the back. On the back of the stand is a brass protractor, marked in degrees, to show the axis at which the rod-shaped box is placed.

The room is partially darkened by drawing down the blinds. The patient is seated in front of the instrument at a distance of about 8 or 10 feet. In a trial-frame he wears a red glass before the right eye, and a green glass before the left. With the right eye he sees nothing but the centre red light; and with the left eye he sees nothing but the two green lights. *The angular distance between the lights is sufficiently great to avoid any tendency to fuse the red light with either of the green lights*, so that the eyes are perfectly dissociated. First the rod-shaped box is put horizontal and the patient is asked whether the three lights appear in the same straight line. If they do, he has no hyperphoria. If the red appears below the level of the green lights, he has right hyperphoria. If it appears above, he has left hyperphoria. The degree of the defect is measured by putting a rotary prism (fig. 27) in the trial-frame and screwing it up until the lights appear in line. Esophoria and exophoria are similarly detected and measured by placing the rod-shaped box vertical—red to the right indicating esophoria, and red to the left, exophoria.

[1] I believe that Mr. Lang was the first to use Snellen's coloured glasses for the measurement of heterophoria. His apparatus is described in "The Methodical Examination of the Eye," page 52.

If one wishes to ascertain the direction of a compound defect, such, for instance, as esophoria with right hyperphoria, the box is rotated until the lights are in the same straight line, though of course not equidistant from each other. The axis is then read off from the protractor at the back of the stand. To ascertain the degree of the compound defect, place the box with its axis at right angles to this, then screw up the rotary prism until the lights are again in the same straight line.

Esophoria.

The liability of an esophoria to cause trouble is determined not so much by its degree as by the condition of the functions of abversion and binocular abduction. A patient who can abvert each eye separately until the cornea touches the outer canthus, and whose binocular abduction is not less than 3°, will as a rule be able to support many degrees of esophoria without inconvenience.

In many cases of esophoria of high degree there is occasional momentary diplopia. When the patient is gazing vacantly without perceiving what is before his eyes, perhaps the visual axes may deviate. Instantly the diplopia awakens the dormant fusion sense, and the eyes immediately recover themselves. These cases are often mistaken for occasional convergent squint. Occasional squints, however, differ from esophoria in that the fusion sense is defective, so that the deviation is not so instantly corrected, and diplopia is either absent or very faint. In an occasional

squint, too, examination with the Maddox rod shows that when the deviation is not actually present there is little or no tendency to convergence. In a case of occasional squint, we have a pair of eyes, not properly controlled by the fusion sense, responding to intermittent and varying nervous impulses. In esophoria there is a constant and definite motor anomaly which is kept in check by a perfect fusion faculty.

Treatment.—Moderate degrees of esophoria never cause inconvenience unless the binocular abduction is very deficient. In these cases the symptoms are relieved by the constant wearing of prisms, apex in, which represent the deficiency of binocular abduction (*not* the degree of esophoria). The prism should be divided between the two eyes. For example, a patient with 5° of esophoria will probably not be inconvenienced thereby. But if his binocular abduction is only 2° instead of 4° he is likely to suffer from "asthenopic" symptoms and occasional diplopia. He will be relieved by wearing prisms, apex in, having a total deviating power of 2°. If the patient already wears glasses for correction of a focal error, the prismatic effect may perhaps be got by decentration of lenses (see Appendix).

In a case of esophoria of high degree requiring treatment, operation is the only resort. If the esophoria is not less then 7° in distant vision, and the near vision test shows the same or a

higher degree, and if abversion and binocular abduction are not markedly below normal, the best procedure is complete central tenotomy of an internal rectus, by the method described on p. 216. If the degree of esophoria is less in near vision than in distant vision, tenotomy is contra-indicated. In a case of esophoria of high degree, with abduction less than 2°, and abversion subnormal, and frequent momentary diplopia, advancement of an external rectus should be performed.

Exophoria.

Uncomplicated exophoria of moderate degree seldom causes any inconvenience. But if, as occasionally happens, there is a defect of dynamic convergence in addition, the patient is likely to suffer from frontal headache, not only after using the eyes in near vision, but often at other times also. In cases of exophoria of high degree the eyes may momentarily deviate, but this is less common than in esophoria.

Treatment.—Prisms are seldom of use in exophoria. Slight cases require no treatment, if uncomplicated. If the case be complicated by convergence deficiency, treatment should be directed to this anomaly. Even if there be no deficiency of dynamic convergence, convergence training often relieves the patient's symptoms, but, in my experience, the effect has been only

transitory. In higher degrees of exophoria, especially if adversion be deficient, the internal rectus muscle should be advanced. One should then aim at producing an operative effect exactly equal to the degree of the exophoria of distant vision, Exophoria is almost invariably due to underaction of the internal recti, scarcely ever to overaction of the externi. For this reason, tenotomy of the rectus externus is not advisable.

Hyperphoria.

Clinically, hyperphoria is the most important of all forms of heterophoria, because of the severity of the symptoms to which it is liable to give rise, and the certainty with which these symptoms may be relieved. The liability of a case to cause trouble depends not only upon the degree of the hyperphoria, but upon the extent of any deficiency of prism-duction in the opposite direction. For instance, an ordinary healthy man who has $\frac{3}{4}°$ of right hyperphoria will probably suffer no inconvenience if the right subduction is as much as 2°: but if it is only 1° or less, he is almost certain to have trouble.

The commonest symptom is frontal headache, coming on towards the end of the day, not especially caused by near work. Some patients complain of giddiness on looking down. Momentary diplopia is not uncommon.

In a marked case of hyperphoria, more often

than not, the palpebral fissure on the hyperphoric side is smaller than that on the other. This asymmetry disappears entirely when the hyperphoria is corrected.

The *treatment*, in any case of hyperphoria of moderate degree, is by prisms to be worn constantly. The prism should be placed, apex up, before the hyperphoric eye. Or, if more than 1° is required, the effect may be divided between the two eyes, the prism before the other eye being placed, of course, apex down. The strength of the prisms should be determined partly by the degree of the hyperphoria and partly by the range of prism duction (see page 179). One should attempt as nearly as possible to correct the hyperphoria and to bring each eye into the middle of the vertical range of prism duction. For example, take a case of right hyperphoria 2°, in which the right superduction (and left subduction) is 3° and the right subduction (and left superduction) is 1°. A prism, apex up, before the right eye, 2° would correct the hyperphoria: but a prism of 1° would suffice to bring the eye into the middle of the range of prism duction. In this case one would order a prism of 1½° as a compromise.

If the hyperphoria is very high—over 4°—operation is usually indicated. If subduction of the hyperphoric eye is not less than 1°, and superduction over 4°, complete central tenotomy of the

superior rectus of this eye is the best procedure. In a case of hyperphoria of very high degree, with very deficient subduction, the inferior rectus muscle of the hyperphoric eye should be advanced.

I have seen a few well-marked cases of so-called *double hyperphoria.* In all there was more or less drooping of both eyelids. A great effort was required to open the eyes widely while looking straight ahead, but in looking up, the lids were lifted normally. Either eye turned up when screened. Subduction of either eye was normal. These cases were possibly due to a faulty nervous connection between the superior recti muscles and the levatores palpebræ superiores. In two of these cases I performed complete central tenotomy of both superior recti, with excellent results.

I have seen several cases of double hyperphoria associated with plus cyclophoria — due probably to underaction of the superior oblique muscles.

I have no note of any case of double kataphoria (downward tendency of each eye).

Cyclophoria.

In order that binocular vision may be possible it is necessary not only that the visual axes of the two eyes shall be directed to the same object, but that their vertical diameters shall be parallel. The work of keeping the vertical diameters

parallel falls almost entirely upon the oblique muscles. If the superior obliques act too feebly, the eyes tend to rotate round a fore-and-aft axis so that their vertical diameters diverge above (plus cyclophoria.) This is very much more common than minus cyclophoria. Plus cyclophoria is not infrequently associated with double hyperphoria.

Cyclophoria may cause nausea, vertigo, and difficulty in judging the true position of the steps in going downstairs.

Not much can be done in the way of direct treatment in cases of cyclophoria. But there are some practical points which deserve careful study.

Close one eye, and hold before the other a strong convex cylindrical lens, axis vertical. Look at a horizontal line, the junction of a floor and ceiling, for instance. The horizontal line still appears horizontal. Now rotate the lens a few degrees. The horizontal line appears to rotate slightly with the lens. In other words, the cylindrical lens rotates the image of the line towards its meridian of greatest convexity.

Next put on a trial-frame and place before each eye a + 1 D. cylindrical lens, axis vertical. Vision will be slightly blurred, but objects will not appear displaced, and the lenses can be worn for a long time without discomfort. Now rotate each lens about 30°, so that their axes diverge above.

On looking down, the floor seems far away, and, on looking up, the ceiling seems quite near—one feels about 7 feet high. Now rotate the cylinders in the opposite direction, so that their axes converge above. The floor appears quite near, and the ceiling high—one feels a dwarf. After a few minutes of this artificial oblique astigmatism, one experiences a feeling of giddiness and nausea, reminiscent of the "giant stride" in one's early school days.

In the light of the former experiment, the explanation is plain. We are accustomed to localise all objects with reference to the horizontal surface (floor, ground, or sea) which supports us. While wearing the convex cylinders with axes divergent above, the images of the horizontal surface of the floor are tilted outwards towards each temple. In order that these images may be received upon corresponding points of the two retinæ, each eye must rotate about a fore-and-aft axis so that the vertical diameters diverge above. This is accomplished by a lessened action of the superior oblique muscles and an increased action of the inferior obliques. But this has also the effect of rotating the two eyes a little upwards. So that, in looking down to the floor, one has to put forth sufficient energy to overcome this upward tendency, as well as to effect the actual downward rotation. And in looking at the ceiling, less than the normal expenditure of energy

is required, owing to the eyes already having an upward tendency. We depend chiefly upon the "muscular sense" of the external ocular muscles in judging the relative positions of objects. Therefore, the increased effort required in looking down, makes the floor appear farther away, and the lessened effort in looking up, makes the ceiling appear lower.

In the experiment with the axes of the cylinders convergent above, the conditions are exactly reversed.

This experiment appears to explain the commonly observed fact that astigmatism is more liable to cause trouble when the axes are oblique than when they are vertical or horizontal. Uncorrected astigmatism, unless the axes in the two eyes are parallel or at right angles, must cause a pseudo-cyclophoria, which should disappear when the astigmatism is corrected.

Now and then one meets with a patient, with oblique astigmatism who is less comfortable with glasses which accurately correct his refractive error than he was without any correction at all. In such a case, one generally finds that he has cyclophoria of an opposite kind to the pseudo-cyclophoria which would be produced by his uncorrected astigmatism. So that they, to a certain extent, neutralised each other. But, with the correction of his astigmatism, the whole of his true cyclophoria becomes manifest. A slight

rotation, say about 5°, of both his cylinders, in the direction which favours the feebly-acting pair of oblique muscles, will often make him quite comfortable without appreciably lowering his visual acuity.

Under certain conditions it is possible, without any rotation of the eyes round a fore and aft axis, to blend images of lines which are slightly tilted in opposite directions (see page 11). Some authors have therefore assumed that the eyes never make an axial rotation in the interests of binocular vision. This view does not accord with clinical and experimental evidence. Fusion of tilted images takes place according to the law stated on page 11.

Rhythmic exercises with prisms, cylinders, &c., are employed by many eminent ophthalmologists in America. I have thoroughly tried all the most approved methods, but have never been able to satisfy myself that I have produced any effect in any case of esophoria or hyperphoria. Cases of exophoria are indirectly benefited by exercising the dynamic convergence, but that is a different matter.

In America there is, no doubt, a tendency to overestimate the importance of small latent deviation tendencies. But this is less harmful than the almost total neglect which the subject meets with in this country.

Here are some examples :—

Mr. R. H., aged 38, a hard worker, and head of a large city business, consulted me on October 17, 1899.

He complained of dull, aching pains in the eyes and forehead. Pain was always relieved by sleep. It was not especially associated with near work. He suffered almost as much during his holidays. His eyes had been examined many times, and he had worn glasses for twelve years. There was a slight drooping of the left upper lid. He was wearing + 2·25 D. sph. each eye. He showed me several prescriptions for glasses, all practically the same. Retinoscopy without mydriatic showed 2 D. of hypermetropia each eye, no astigmatism. His corrected vision was $\frac{6}{6}$ easily, each eye. On investigating the motor balance of his eyes, I found he had nearly 2° left hyperphoria. Left superduction and right subduction were each 4° ; and left subduction and right superduction were each 1°. I ordered spectacles + 2 D. sph. each eye, the right lens to be combined with a $\frac{3}{4}°$ prism, apex down, and the left lens with a similar prism, apex up.

On September 26, 1902, three years later, I saw the patient again. He has worn the glasses constantly, with perfect comfort, and is entirely free from the old trouble.

Miss E. B., aged 33, seen with Mr. Devereux Marshall on February 6, 1903. Patient had suffered from frontal headache and occasional diplopia for as long as she could remember. Headaches were always relieved by sleep, were independent of occupation, and became more severe towards the end of the day. She had for many years worn an exact correction of her ametropia (+ 0·75 D. cyl. ax. vert. each eye), but the glasses gave her no relief.

On investigating the motor balance of the eyes we found left hyperphoria 4°, exophoria 1°. Right superduction and left subduction barely 1°. Right subduction and left superduction each 6°. We ordered, for constant wear, to be incorporated with her cylinders, R. E. prisms $1\frac{1}{2}°$, apex down, L. E. prism $1\frac{1}{2}°$, apex up.

The patient has been seen twice since that date. She is perfectly comfortable and entirely free from headaches.

Miss F. L., aged 17, was brought to me on February 2, 1900. She was wearing spectacles + 1 D. sph. She said that when she was tired her eyes would often be crossed for a moment and she then would see double. The crossing of the eyes only lasted an instant. She had frequent frontal headaches. She had worn glasses since she was ten years of age. She had been told that she suffered from "periodic strabismus." Retinoscopy without mydriatic showed hypermetropia of 0·5 D. only. Vision of each eye $\frac{6}{5}$. Examination with the Maddox rod and tangent scale showed esophoria, 9°, in distant vision. Esophoria, in near vision, was 8°. Binocular prism abduction was only 2°. R. E. could be abverted until the cornea touched the outer canthus. L. E. abversion not quite so complete. The fusion faculty was perfect. The case was not one of "periodic strabismus" at all, but an example of esophoria of high degree.

February 5, 1900.—Retinoscopy was confirmed under atropine.

February 20, 1900.—I advanced the left external rectus muscle (of course without tenotomising the internus).

February 27, 1900.—Stitches removed.

March 29, 1900.—Patient has 1° esophoria in distant vision, perfect orthophoria in near vision, binocular prism abduction 5°. Patient never has diplopia now.

June 18, 1902.—Patient has not been seen to cross her eyes since the operation, and she has had no diplopia. She is quite free from headaches now. She has, of course, not worn glasses since the operation.

Captain P., aged about 30, consulted me on July 11, 1899. He had always suffered a good deal from head-

aches, and had had occasional momentary diplopia, but had never been told that he squinted. Both the headaches and the diplopia had been more frequent since he had "fever" in India four years previously. He had had his eyes examined several times, and had once been ordered reading glasses, but they had done no good.

Retinoscopy showed that he had no notable refractive error. On examining the motor balance of his eyes, he was found to have left hyperphoria $2\frac{1}{2}°$. His right superduction and left superduction were each scarcely $1°$. His right subduction and left superduction were each $5°$. Excursions of each eye separately were quite full.

August 3, 1899.—On repeating the examination, I got precisely the same results. As there was no refractive error which would necessitate glasses, I did not wish to burden him with prisms, I therefore advised operation.

August 14, 1899.—I performed complete central tenotomy of the left superior rectus, after the method described on page 216. The after treatment consisted only of a shell and a pad of gauze worn over the L. E. for a few days, and bathing with boric lotion.

August 29, 1899.—Wound healed and eyes perfectly comfortable. There is now right hyperphoria of less than $\frac{1}{2}°$.

July 11, 1902.—Patient has been free from headaches and the occasional diplopia since the operation. There is now no measureable degree of heterophoria. The streak of light with the rod before the R. E. is at the upper part of the flame, showing left hyperphoria of less than $\frac{1}{4}°$.

Mr. S. H.—I saw quite recently an exceedingly instructive case, a gentleman aged 20, sent me by Dr. Bolton Tomson. From early childhood until one year ago, the patient had suffered from severe head-

aches coming on towards the end of the day. He had occasional diplopia, one image being over the other. He also had frequent attacks of typical migraine. During the last year, he has seen double constantly, and the right eye has squinted downwards. He can still, by a great effort, overcome the deviation and blend the images. The diplopia is so intense that he is only comfortable when one or other eye is covered. But *since the hyperphoria gave place to an actual deviation, the headaches and migraine have completely disappeared.*

There is no important refractive error. R. E. deviates downwards (or L. E. upwards) 8°. Either eye deviates outwards 5°. Prisms of this strength give binocular vision with orthophoria.

I propose to advance the L. inferior rectus muscle, which I have no doubt will result in cure of the whole trouble. If this operation had been performed many years ago the patient would have been spared much unnecessary suffering.

Insufficiency of Dynamic Convergence.

This is not a heterophoria, but it is convenient to discuss it in this chapter.

There has been much confusion on the subject of insufficiency of convergence, because authors have not clearly distinguished between static and dynamic convergence.

A person whose visual apparatus is normal has no static convergence at any time ; in distant vision he exercises no dynamic convergence ; in near vision his dynamic convergence exactly suffices to cause both visual axes to be directed to the near object. If his dynamic convergence

were excessive, there would be a *tendency* to convergent squint in near vision; if it were insufficient, he would have difficulty in maintaining convergence, as in reading, for instance, for any length of time.

A person who has a convergent squint, which persists after correction of any refractive error, has an unchecked static convergence; one who has esophoria has a static convergence which is kept in check by constant muscular effort,[1] evoked by the desire for binocular vision. Static convergence is a minus quantity in divergent squint. In exophoria there is a minus static convergence which is neutralised by an abnormal effort of dynamic convergence. In these cases the power of dynamic convergence may or may not be normal.

A patient, therefore, who has exophoria in distant vision and the same degree of exophoria, or less, in near vision, cannot properly be said to have insufficiency of convergence. This distinction between exophoria and insufficiency of dynamic convergence is of supreme practical importance, because the treatment of the two affections differs entirely. In the former case, if any treatment be required at all, operation is usually necessary; in the latter, operation is al-

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[1] This "without prejudice" to the much-debated question of the existence of a cerebral centre for divergence.

ways contra-indicated, and benefit may often be obtained from exercises.

Insufficiency of dynamic convergence, apart from neuropathic cases, is not common.

The symptoms produced by insufficiency of convergence are pain in the brow after reading, and a tendency to hold the book at a long distance from the eyes (apart from any error of static or dynamic refraction).

Convergence being a voluntary act, the extreme degree of which any individual is capable will vary from time to time, according to the state of his health and the amount of energy he is able to put forth at the moment. Elaborate instruments for determining the near point of convergence are, therefore, not required. Moreover, this information is not of much practical use.

The best procedure is to test the horizontal motor balance of the eyes, first in distant vision, and then at ten inches. If there is no more exophoria, or no less exophoria, in near vision than there is in distant vision, the patient has no insufficiency of convergence. If the patient has orthophoria in distant vision and exophoria in near vision, or if there is more exophoria, or less esophoria, in near than in distant vision, he has insufficiency of dynamic convergence of a degree equal to the difference.

In an uncomplicated case of insufficiency of convergence, exercises should be tried. The

following procedure is as good as any :—Any error of static or dynamic refraction is corrected by glasses. The patient begins reading a book at the ordinary distance. Then, while still reading, he gradually brings the book nearer his eyes until the print begins to be blurred. He then slowly removes the book to the ordinary reading distance. This is repeated. At about every tenth line he looks into the distance for a moment, in order to completely relax his convergence. Two or three pages should be read in this way, three or four times a day for a month. This simple plan has given quite as good results as the more elaborate methods which I have tried. This is perhaps because the patient finds it more convenient to carry out, no special apparatus being required.

It has been objected that the accommodation is exercised at the same time as the convergence. I do not think that it would be advisable, in young subjects at any rate, entirely to dissociate dynamic convergence from the effort of accommodation with which it is normally always associated. But if it should seem advisable to relieve the strain on the accommodation during the exercises, this may be done in either of two ways—the patient may wear convex glasses so as to exercise less accommodation with a given amount of convergence, or, what comes to the same thing, he may wear prisms, apex in, so as to exercise more convergence with a given amount of accommodation.

These rhythmic exercises do not increase the power of the ocular muscles (any more than voice

training increases the power of the laryngeal muscles), so they do not in the least diminish exophoria in distant vision. But they often much improve the power of dynamic convergence, by teaching the nervous apparatus to respond more readily to the will.

In a case in which exercises have failed and the symptoms are troublesome one may, as a pis aller, order prisms, apices out, for near vision.

CHAPTER XII.

OPERATIONS ON THE EXTERNAL OCULAR MUSCLES.

THE operations commonly performed on the muscles of the eye are advancement and tenotomy. Either of these measures may be employed alone, or advancement of one muscle may be combined with tenotomy of its opponent.

The indications for these operations are fully discussed in previous chapters.

ADVANCEMENT.

Very many different methods of advancement of a rectus muscle have been described. I have tried many of these repeatedly, also two methods of shortening the tendon by folding it upon itself. None of these has proved entirely satisfactory. With practice, one can always make sure of inserting the sutures firmly in the tough fibrous tissue near the margin of the cornea, without putting in the needle dangerously deeply. The difficulty is with the end of the sutures attached to the muscle. I found that two of these methods of operating gave better results than the others. In one operation, three or four sutures are simply

passed through the tough circumcorneal tissue, and through the muscle, and tied. If these sutures hold for a week, the results are permanent. But the muscle end of the suture often cuts its way through in three or four days. And in any case, one has to produce an over effect at the time of the operation, to allow for slacking of these sutures, so that precision is not attainable by this method. In the other operation, the sutures are knotted on the muscle, either in one or two parts. This nearly always gives good *immediate* results, but a very large proportion of the cases relapse within a few weeks. This is probably due to the fact that the muscle atrophies in front of the ligatures, and so is only attached to the globe by its lateral expansions, which subsequently stretch.

I devised the following operation with the object of combining the advantages and avoiding the disadvantages of the two methods to which I have just referred.

The Author's Advancement Operation.

In this operation a firm, unyielding hold is got for the sutures at each end, so that any desired degree of rotation of the eyeball may be produced. For moderate deviations, I advance a muscle without tenotomising its opponent. For squints of very high degree, I usually first tenotomise the opposing muscle to avoid refraction of the

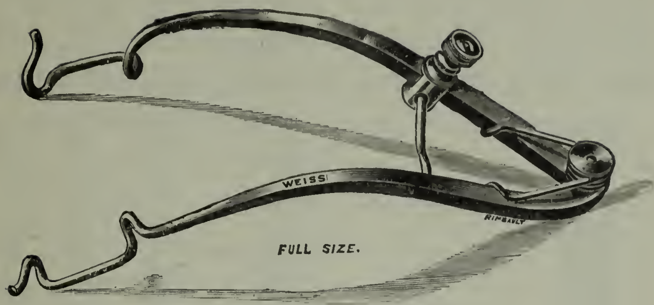

FIG. 29.

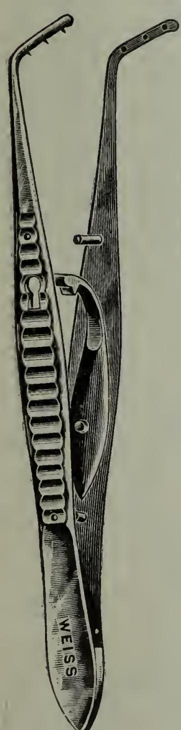

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FIG. 30.

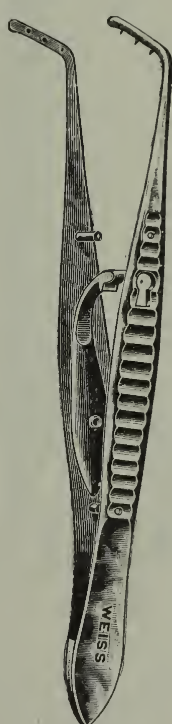

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FIG. 31.

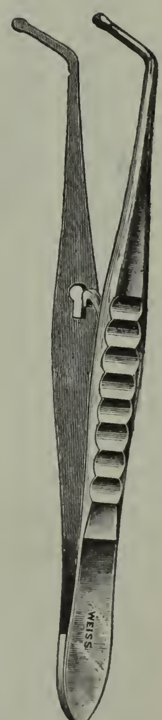

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FIG. 32.

globe. The anatomical relations of the advanced muscle are disturbed as little as possible. As the middle part of the muscle is not included

FIG. 33.

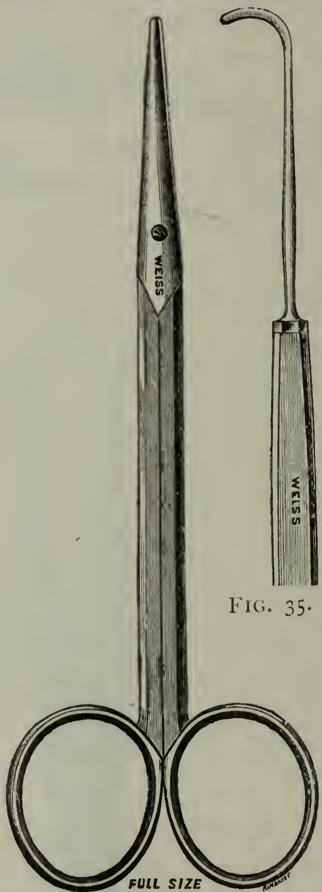


FIG. 35.

FIG. 34.

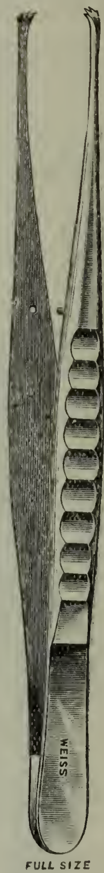


FIG. 36.

in the sutures, its main *blood supply is not interfered with.* The immediate effect produced is the final result.

Instruments.—The ordinary advancement instruments are required: Speculum (fig. 29), straight blunt-pointed scissors (fig. 34), fixation forceps with projecting teeth which take a firm hold of the sclerotic (fig. 36), Prince's advancement forceps, right and left (figs. 30 and 31), or the smooth probe-pointed forceps (fig. 32), needle holder (fig. 33), needles and thread; and if a tenotomy is to be done at the same time, a tenotomy hook also (fig. 35). The needles I use are made for me by Messrs. Weiss and Son. They are small, curved needles with large eyes. They must be very sharp. The jaws of the needle holder which I use (fig. 33), are curved transversely to fit these needles. Flat jaws, however small, are apt to break a curved needle. The advancement forceps shown in fig. 32, being smooth and probe-pointed, is easier to introduce than the ordinary form of Prince's. Both blades being alike, only one forceps is required instead of two. The spring is strong so that the smooth blades show no tendency to slip under any strain to which one ought to subject the tissues.

The blunt instruments are sterilised by boiling for five or ten minutes in water containing a little washing soda. The scissors are boiled for about a minute. The eyes of new needles are cleared with the point of a sewing needle. The needles are then boiled for not more than a minute, before being threaded. I used to use only new needles,

to ensure their being as sharp as possible. I find, however, that it is quite easy to sharpen them on a fine oiled stone : even new needles can be much improved. They should then be kept in a flannel book ; the paper in which they are sold is apt to injure delicate points.

The suture material is a very important detail. It should be thick. The thin stuff sold as "eye silk" cuts like a knife. I used thick black silk prepared as follows: A reel of the silk is wound loosely round a winder (made by bending up a piece of wire). It is boiled in water, to sterilise it and to remove the superfluous colouring matter. It is then dried before a fire. The end of the silk is then threaded through a large glass bead. The glass bead is then dropped into a glass beaker containing a very hot mixture of white beeswax, three parts, and white vaseline, five parts. The whole of the silk is drawn through the boiling mixture, and is wound on a large glass reel. It is kept in a sterilised glass jar, always ready for use without further preparation.

In preparing for the operation, a piece of the silk, about a foot long, is drawn out of the jar with sterilised forceps; it is threaded through the sterilised needle as far as its middle. The two halves of the waxed silk are then twisted together into a single cord. Two of these threaded needles are required. The part of the suture which meets

with the greatest resistance in passing through the tissues, is that near the eye of the needle. The thread must be double here in any case, so one may as well have the benefit of the double thickness throughout. The wax-soaked thread is sufficiently stiff, at the ordinary temperature of the air, to abstain from tying itself into undesired knots during the operation, but at the body temperature it is quite supple. It glides easily and with a minimum of damage to the tissues—like a well greased catheter. It is practically non-absorbent—an important point in a region which cannot be absolutely aseptic.

Anæsthesia.—For young children a general anæsthetic, preferably chloroform, is necessary. For older patients a local anæsthetic is sufficient. I use the crystals of hydrochlorate of cocaine, applied twice before the operation at intervals of about five minutes. During general anæsthesia the eyes usually diverge, so that one has to remember the angle of the squint and produce approximately the effect required. Under a local anæsthetic there is no such divergence, so that one can gauge exactly the extent to which the muscle is to be advanced. For this reason, I never operate under general anæsthesia unless I can rely upon a trained fusion faculty to do the fine adjustment.

The operation.—The hands of the surgeon and nurses are sterilised. The patient's face is

thoroughly cleansed. The conjunctival sac is irrigated with sterilised saline solution (60 grains to a pint of water). If the lacrymal apparatus and conjunctiva are not perfectly healthy, the operation is postponed until they are. Just after the irrigation, and from time to time during the operation, a few drops of supra-renal gland extract are instilled. I use Parke Davis and Co.'s adrenalin chloride solution. The vascular constriction caused by the extract makes the operation bloodless or nearly so, so that one is not impeded in one's work by the necessity for constant sponging. The patient lies on the table in a good light, with his feet towards the window. His lids are held open by the speculum. The surgeon, standing behind the patient's head, grasps the conjunctiva with the toothed forceps, while, with the scissors, he makes a straight vertical incision through it about half an inch long. The middle of the incision is close to the corneal margin. A similar incision is then made through the capsule of Tenon. The conjunctiva and capsule then retract, or, if necessary, they are pushed back, so as to expose the insertion of the tendon. If the angle of the squint is of high degree, the vertical incision through the membranes is made curved instead of straight, the convexity of the curve being towards the cornea. This is to allow the membranes to retract more freely. One blade of the advancement forceps is now passed under the

tendon, after the manner of a tenotomy hook, the other blade being superficial to the conjunctiva. The forceps is now closed, so that tendon, capsule of Tenon, and conjunctiva are all firmly clamped together, with their relations undisturbed except for the retraction of the membranes. The tendon, and a few little fibrous bands beneath the tendon, are now divided with scissors, at their insertion into the sclerotic. The advancement forceps, holding the tendon, capsule, and conjunctiva, can now easily be lifted up so as to get a good view of the under side of the muscle.

One of the needles is then passed inwards at A, through conjunctiva, capsule, and muscle. It is then again passed through muscle, capsule, and conjunctiva, and brought out at B. The bight of the thread thus encloses about the lower fourth of the width of the muscle, together with its tendinous expansions and capsule and conjunctiva. The other needle is similarly entered at A′, passed through conjunctiva, capsule, and muscle, and brought out at the under side of the muscle. It is then entered again at the under side of the muscle and brought out through the conjunctiva at B′, the bight of this suture thus enclosing the upper fourth of the width of the muscle, &c. The object of inserting both sutures, before proceeding further with either, is that they may be symmetrically placed. The ends of the thread from A′ and B′ are then knotted tightly

at C. The end bearing the needle is then entered at D, and passed through conjunctiva, capsule, and muscle, and carried beneath the lower blade of the advancement forceps nearly to the corneal margin. The needle is here passed

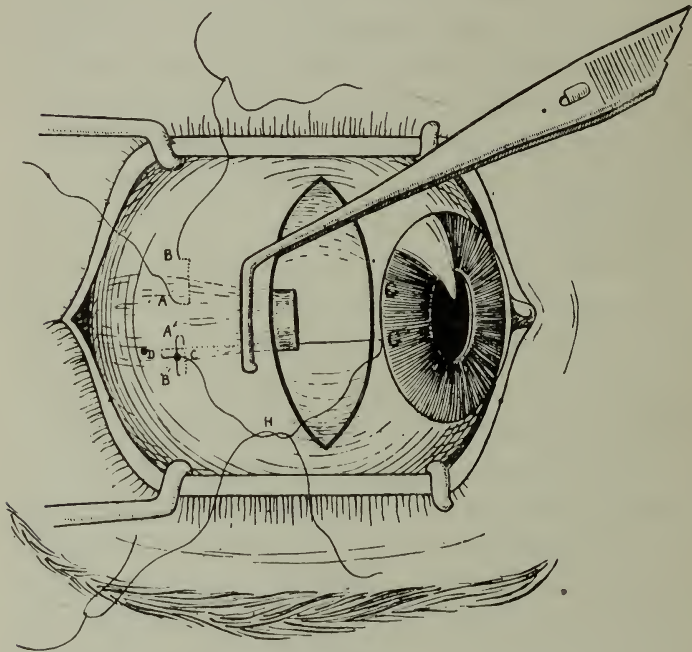


FIG. 37.

through the tough circumcorneal fibrous tissue, and brought out at G′. The two ends of the thread are then temporarily tied loosely, with a single hitch, at H. The first suture is then similarly completed. The anterior part of the muscle and capsule and conjunctiva are then

removed, by cutting them through with scissors behind where they are grasped by the advancement forceps. The gap is then closed by tightening and securely tying each suture at H H, so that the eyeball is rotated in its correct position, and the anterior end of the muscle is brought nearly up to the corneal margin at GG′.

In operating under cocaine, before the knots are tied at H H, an assistant holds the globe in the primary position with forceps, while the patient is told to try to look away from the operated muscle. This relaxes the muscle while it is being drawn forward by the sutures. The sutures are then temporarily secured at H H, by the first hitch of "the surgeon's knot." The assistant then releases the globe. The fine adjustment is done by tightening or loosening the hitches at H H, the result being checked by the mirror test or by the reflection of a candle flame on the corneæ. The surgeon's knots at H H are then completed.

The longitudinal position, on the muscle, of the loops A B C and A′ B′ C′, varies approximately according to the degree of rotation required.

In operating under cocaine, the immediate effect is the permanent result. No over-correction, therefore, is necessary. In operating under general anæsthesia, one has to bear in mind the angle of the deviation and produce approximately that degree of rotation.

After the operation, the eye is irrigated with sterilised saline solution, a little boric ointment smeared on the edges of the lids, and a gauze pad applied.

After-treatment.—The eye is irrigated and dressed every day. The more quiet the eye is kept, during the first few days, the more quickly will the wound heal. I prefer, therefore, to keep the patient in bed with both eyes bandaged for the first four or five days. After this time his unoperated eye is uncovered, and atropised to prevent any effort of accommodation. His spectacles, if he has any, are put on over his bandage. It is a good plan to stick two pieces of postage stamp paper on the lens before the unoperated eye, so as to leave a narrow vertical slit between them. If the patient looks only through the slit, he can look up or down, but does not make any horizontal or accommodative movements, which might retard the healing of the wound. The stitches are removed on the eighth day. The bandage is discarded two days later.

Musculo-capsular advancement.—In many cases of neuropathic divergence, and in some old cases of divergence following tenotomy of an internal rectus, it is advisable to bring forward the capsule of Tenon and conjunctiva as well as the enfeebled muscle. These membranes have little elasticity, so that movement in the opposite direction will be restricted.

Seize the conjunctiva and capsule of Tenon just above or below the insertion of the muscle to be advanced. Snip through these membranes with scissors. Introduce one blade of an advancement forceps, and hook this blade under the tendon, after the manner of a tenotomy hook. Close the forceps. The membranes and tendon are thus clamped together, so that no retraction of the former is permitted. Now make the long vertical incision near the corneal margin, and proceed with the advancement as already described.

Secondary advancement.—Not infrequently a patient presents himself with one eye widely divergent as a result of tenotomy of an internal rectus muscle. Usually, an excellent cosmetic result may be obtained by advancing the retracted muscle, even after many years. Sometimes, however, the muscle is much atrophied from disuse.

The conjunctiva and capsule of Tenon will be matted to the globe in the neighbourhood of the former insertion of the muscle, or the eyeball may be quite destitute of covering in this region. Grasp the membranes, with toothed forceps, well above or below the scarred area, and, with the scissors, separate them from the nasal side of the eye. A squint hook may be of assistance in this. If the muscle is not found attached to some portion of the anterior segment of the globe it is of no use searching for it with a squint hook far back. It is never found attached to the posterior

15

hemisphere. Seize the coverings of the eye in the region of the sunken caruncle, pull them forward, lift them up. The muscle, or what remains of it, will be seen on their under surface. I think that, after tenotomy, the tendon fails to become reattached directly to the globe far more often than is generally supposed. If the muscle be fairly good it may be advanced in the ordinary way. If, however, it be much atrophied, a musculo-capsular advancement is preferable. The muscle and membranes are seized with toothed forceps and drawn between the jaws of an advancement forceps. The surface to which the muscle and membranes are to be attached must be well refreshed.

Tenotomy.

There are several slightly different methods of performing this little operation. The following is as good as any other. The instruments required are speculum (fig. 29), straight blunt-pointed scissors (fig. 34), tenotomy hook (fig. 35), and fixation forceps (fig. 36). The patient lies on a table. Both eyes are cocainised. The eye to be operated upon is irrigated with sterilised saline solution, and a drop of supra-renal extract is instilled. In operating on the left internal rectus, it is more convenient to stand in front, and on the left of the patient. In tenotomising any of the other recti, I prefer to stand behind the patient's head. The speculum is inserted, if the muscle to

be tenotomised be one of the external or internal recti. In the case of a superior or inferior rectus, the lids should be held open by the fingers of an assistant. Tell the patient to look in a direction opposite to that of the tendon to be divided, so as to bring its insertion well forward. With the forceps, pick up the conjunctiva over the insertion of the tendon, and, with the scissors, make an incision, about one-third of an inch long, in a direction at right angles to that of the tendon. Now divide the capsule of Tenon in the same way. This brings the insertion of the tendon into view. While the forceps still hold up the cut edge of the capsule, make a few short snips with the scissors near one border of the tendon, until the point of the scissors is felt to slip freely back without encountering any resistance. Now lay down the scissors, and take up the hook in the right hand. Pass the point of the hook into this incision, and hook it round the insertion of the tendon, until it appears at the other border. During this manœuvre, the point of the hook should be kept in contact with the sclerotic. Now lay down the forceps, and transfer the hook to the left hand. Take care to avoid any dragging with the hook, as this causes pain. With the scissors, cut between the point of the hook and the globe, until the tendon is divided at its insertion, and the hook comes away. It is usual to reintroduce the hook, to seek for any fibres of insertion which

may have escaped division. It is not necessary or advisable to suture the conjunctiva, unless the conjunctival incision is unusually large.

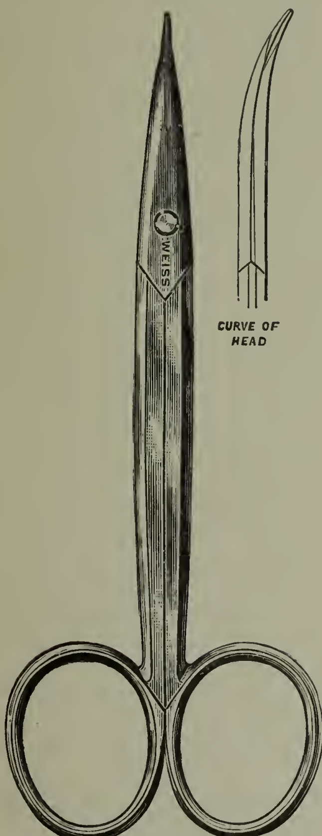
After the operation, there is a very considerable defect of movement in the direction of action of the tenotomised muscle. This, to some extent, subsequently disappears. The *average* effect of a tenotomy of the internal rectus is 13°, and of the other recti less than half this amount. But it varies within extremely wide limits.

A pad and bandage should be worn for the first forty-eight hours, after which it may be discarded. The eye should be bathed with boric lotion, three or four times a day, until the wound is healed.

Complete central tenotomy.—In America, partial tenotomies are very commonly performed. I believe these to be useless, as, until the whole of the tendon proper has been divided, no appreciable effect is produced. But the neat and precise method by which these partial tenotomies are performed is very well worth copying. By this method, the tendon itself may be completely divided, leaving its lateral expansions absolutely intact.

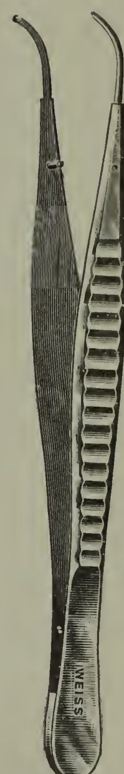
The patient is prepared as for an ordinary tenotomy. The forceps, scissors, and tenotomy hook required are those of Stevens (figs. 40, 38, and 39). The surgeon, standing behind the patient's head, seizes, with the forceps, the conjunctiva over the tendon near its insertion. A transverse incision

of sufficient length is made through the conjunctiva. The capsule of Tenon is now similarly incised, to an extent just sufficient to expose the



FULL SIZE
FIG. 38.

FULL SIZE
FIG. 39.



FULL SIZE
FIG. 40.

tendon. The central fibres of the tendon are next grasped by the forceps, and a button-hole is snipped through the tendon with the scissors. The forceps being laid aside, the small tenotomy

hook is introduced through this button-hole, with its point turned towards one border of the tendon, Half the tendon is cautiously snipped through on the hook, from the centre towards the edge. The point of the hook is now turned round, and the other half of the tendon similarly divided.

When the hook is first introduced through the button-hole, on lifting it up, the tension of the tendon is felt. When the whole of the tendon proper has been divided, considerable tension is still felt, by means of the hook, at the edges of the wound. This resistance is due to the lateral expansions of the tendon. These should on no account be divided, as it is upon the preservation of these lateral expansions that the safety of this operation depends. Sometimes it is impossible to define exactly the edge of the tendon, owing to its gradually merging into its lateral expansions. In such a case, one has to be guided by the degree of tension felt by the hook.

No after-treatment is required beyond frequent bathing with boric lotion or sterilised saline solution.

This operation appears to be a safe one when judiciously employed, but the effect produced by it is small—about 6° or 7° in the case of the internal rectus, and 3° in the case of the superior rectus. It varies very little in different cases, and shows no tendency to increase with time. I have usually seen a slight decrease after a few weeks.

APPENDIX.

Congenital Amblyopia.

Table VI. shows the degrees of refractive error and the visual acuity in the twenty-three cases of congenital amblyopia described in Chapter V.

Table VI.

Better Eye.			Worse Eye.		
Refractive error.		Vision.	Refractive error.		Vision.
Lower meridian.	Higher meridian.		Lower meridian.	Higher meridian.	
+ 1	+ 1	6/6	+ 2·5	+ 4	6/60
— 0·5	0	6/6	— 0·5	+ 4·5	6/36
+ 1·5	+ 1·5	6/6	+ 1	+ 3·5	6/24
— 1·5	+ 2	6/9	— 2	+ 2·5	6/18
+ 2	+ 1	6/6	+ 2	+ 5	6/18
+ 2	+ 1·25	6/6	0	+ 5·25	6/36
+ 1·25	+ 1·25	6/6	+ 5	+ 7	6/36
+ 1·5	+ 1·5	6/6	+ 5	+ 6·5	6/24
+ 4	+ 4	6/6	+ 5·5	+ 8·5	6/24
+ 0·5	+ 0·5	6/6	+ 1	+ 1	6/36
+ 0·5	+ 0·5	6/6	+ 4	+ 6	6/60
+ 1	+ 1·25	6/6	+ 1	+ 4·5	6/24
+ 3·5	+ 3·5	6/6	+ 2	+ 6·5	6/36
+ 1·5	+ 1·75	6/6	+ 1·5	+ 4·75	6/18
+ 0·5	+ 0·5	6/6	— 1	+ 3	6/24
+ 1	+ 1	6/6	+ 1	+ 4	6/24
0	0	6/6	+ 4	+ 6·5	6/24
+ 3	+ 3·5	6/9	+ 3	+ 7	6/36
+ 1	+ 1	6/6	— 1·5	+ 2·5	6/24
+ 0·75	+ 1	6/6	+ 3	+ 5·5	6/18
+ 2	+ 2	6/6	+ 2	+ 6·5	6/36
+ 0·5	+ 0·5	6/6	+ 1	+ 4·5	6/18
+ 0·25	+ 0·75	6/6	+ 1·5	+ 5·5	6/24

Table VII. shows the refractive error and the visual acuity of the eleven cases in Table III., Chapter V., which had an amblyopia of $\frac{6}{18}$ or higher. The visual defect in these cases also is almost certainly congenital.

Table VII.

Fixing Eye.			Deviating Eye.		
Refractive error.		Vision.	Refractive error.		Vision.
Lower Meridian.	Higher Meridian.		Higher Meridian.	Lower Meridian.	
+ 2	+ 2	$\frac{6}{6}$	+ 1·5	+ 5·5	$\frac{6}{24}$
+ 3·5	+ 4	$\frac{6}{6}$	+ 3·5	+ 6	$\frac{6}{18}$
+ 1·5	+ 1·5	$\frac{6}{6}$	+ 1·5	+ 5	$\frac{6}{24}$
+ 4	+ 4·5	$\frac{6}{6}$	+ 2	+ 5·25	$\frac{6}{18}$
+ 2	+ 3	$\frac{6}{6}$	+ 2	+ 7	$\frac{6}{36}$
+ 1	+ 1	$\frac{6}{6}$	+ 1	+ 3·5	$\frac{6}{18}$
+ 2·5	+ 3	?	+ 0·5	+ 3·5	$\frac{6}{18}$
+ 3	+ 3	$\frac{6}{6}$	+ 3·5	+ 7·5	$\frac{6}{60}$
+ 0·5	+ 0·5	$\frac{6}{6}$	— 1	+ 2·75	$\frac{6}{18}$
+ 2·75	+ 3·5	$\frac{6}{6}$	+ 1	+ 5·5	$\frac{6}{24}$
+ 1·25	+ 1·25	$\frac{6}{6}$	+ 4·5	+ 8·5	$\frac{6}{24}$

Prisms and Decentred Lenses.

There are several systems of numbering prisms. In ordering a prism it is necessary, therefore, to specify which system one uses. In this book the strength of a prism is expressed by the number of degrees which it deflects a ray of light. This "deviating power" is about half the geometrical angle (the angle between the two plane surfaces).

A prism causes mal-projection, and chromatic dispersion of white light. Clinically, mal-projection is avoided by dividing the prism between the two eyes. For instance, in a case of right hyperphoria 2°, if one wished to correct $1\frac{1}{2}$° of the error, one would order a prism $\frac{3}{4}$° apex up, before the right eye, and a prism of the same strength, apex down, before the left eye.

Chromatic dispersion is not noticeable in a prism which does not exceed 2° deviating power.

A pencil of parallel rays which traverses a spherical lens at its optical centre, is made convergent or divergent. A pencil of rays, traversing a lens towards its periphery, is deflected in addition (as by a prism) towards the axis of a convex lens away from the axis of a concave lens.

Glasses containing prisms in combination with spherical and cylindrical lenses are expensive, because they must be specially ground—the optician cannot prepare them from his stock. But if the patient has to wear fairly strong lenses, and if the required prismatic effect is small, this may be secured by decentring the spectacle glass. That is, the optician, instead of cutting the spectacle glass from the middle of one of his ready-ground lenses, cuts it from one side. This is much cheaper. The effect of decentring a cylindrical lens, in a direction at right angles to its axis, is the same as that of decentring a spherical lens. In a lens of the ordinary stock size, there is room to decentre a medium-sized spectacle glass about 3 mm., *i.e.*, a total of 6 mm. in the two eyes.

The following table, prepared from Dr. Maddox's formula, shows the prismatic effect of decentring lenses.

	2 mm.	3 mm.	4 mm.	5 mm.	6 mm.
2 D.	14′	21′	27′	35′	41′
3 D.	21′	31′	41′	52′	1° 2′
4 D.	27′	41′	55′	1° 10′	1° 22′
5 D.	35′	52′	1° 10′	1° 26′	1° 43′
6 D.	41′	1° 2′	1° 22′	1° 43′	2° 4′
7 D.	48′	1° 12′	1° 36′	2°	2° 24′
8 D	55′	1° 22′	1° 50′	2° 19′	2° 45′

Results of Fusion Training.

In the earlier cases I had not perfected my methods of fusion training, and in recent cases sufficient time has not yet elapsed to show that the results are permanent. I therefore give the results of fusion training in 100 consecutive cases, beginning January, 1896. Five were what I have called essentially alternating squints, in which I was unable to get even simultaneous vision of the two object slides. In 17 cases the patients were more than 6 years of age. I got a moderately good result in two of these, and failed in the other 15. I scarcely ever now attempt fusion training after 6 years of age. Of the remaining 78 cases, which alone were suitable for fusion training, I failed in 12 cases (owing to intractability of patient, irregularity of attendance, or apparent absence of the power acquiring fusion) ; in seven cases the result was only moderate, and in 59 cases a good amplitude of fusion was developed. In two of these cases the parents refused advancement which was necessary to complete the cure ; four cases have been lost sight of ; the remaining 53 cases are perfectly and permanently cured.

Results of Advancement Operations.

In performing advancement I have, since 1898, relied *exclusively* upon the method described in Chapter XII. I therefore give results of 100 consecutive advancement operations, beginning January, 1899. I have re-examined most of these cases within the present year (1906).

By *primary advancement* I mean advancement of a muscle which had not previously been operated upon. In some of these cases the opposing muscle had previously been tenotomised by some other surgeon. By *secondary advancement* I mean operation

upon a muscle which had previously been tenotomised or unsuccessfully advanced.

In cases in which the fusion sense has been fairly well developed, but in which the deviation persists, the object of operation is to put the eyes into such a position that the patient will have binocular single vision. If the fusion sense cannot be developed, one aims at removing the visible deformity. Cases which come up to this standard I have classified as successful. Those which do not I have called unsuccessful, although in all these cases there was considerable improvement.

Eighty-six operations were performed under cocaine and 14 under chloroform.

The 100 cases consisted of :—

Primary	advancement of	external	rectus	...	77
,,	,,	internal	,,	...	8
,,	,,	inferior	,,	...	1
Secondary	,,	external	,,	...	4
,,	,,	internal	,,	...	9
,,	,,	superior	,,	...	1

The 77 cases of primary advancement of external rectus included one case of congenital paralysis of external rectus and two cases in which the muscle was atrophied. In these three cases, though a perfect result was not to be expected, musculo-capsular advancement, combined with tenotomy of the opposing muscle, produced considerable improvement in the appearance.

Of the 74 remaining primary advancements of external rectus, 66 were successful ; in three other cases a second operation[1] proved successful ; in one a second operation was recommended but was refused ; in four cases, though the results did not satisfy me,

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[1] I have not included these three cases in the list of secondary advancements recorded below.

the patients were quite satisfied, so, as there was no fusion sense in any of these four cases, I did not urge further operation. In 17 of these 74 cases the internal rectus was tenotomised at the same time.

Of the eight primary advancements of internal rectus, two were old myopic divergent squints, and two were neuropathic divergent squints with some fusion sense. These were successful. The remaining four were neuropathic divergent squints with no fusion sense. Considerable improvement in appearance was produced in these four cases, but the results were far from perfect (see p. 138). The external rectus was not tenotomised in any case.

The primary advancement of inferior rectus was in a case of vertical deviation of high degree with absence of fusion sense. I succeeded in removing the deformity.

The four secondary advancements of external rectus were in cases in which some one had unsuccessfully attempted advancement. In one case I succeeded. In the other three the previous mutilation[1] of the parts rendered a perfect result impossible.

The nine secondary advancements of the internal rectus were all cases in which tenotomy of this muscle had been followed by divergence of the eye. I was responsible for two of these tenotomies. Five of these secondary advancements were successful ; in three the condition was improved ; in one there was no improvement.

The secondary advancement of the superior rectus has already been described (Case D, 227, p. 153).

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[1] If a suture has been tied on the muscle, *including its whole width*, the muscle in front of the ligature atrophies just as surely as if it had been cut off with scissors.

If a surgeon in advancing a muscle has isolated it from its overlying membranes and its lateral expansions, if one has to operate again upon this muscle one finds a shapeless mat of muscle and scar tissue.

The Deviometer (p. 89).

The patient's eye is only two feet from the instrument. In looking at the button above the light he will, therefore, exercise a dynamic convergence proportionate to that distance. It has often been suggested to me that this would cause the degree of a convergent squint to appear greater than it really is. This is not the case, because the surgeon's eye is at the same distance, just above the zero of the scale. This is easily demonstrated by experiment with a normal-sighted person. When he looks at the button both visual axes converge to this point. The surgeon places his eye at the same distance—just above the button—so that he sees the vertical lines of light occupying symmetrical positions on the corneæ of the observed person, showing that the latter has no squint. If the observed person could look at the button with one eye, without exercising any dynamic convergence (keeping his visual axes parallel), he would appear to the surgeon to have a divergent squint—in fact, he *would* have a divergent squint for that distance.

No adjustments are required except that the 60 cm. string must be kept taut. As the scale is flat, instead of a curved arc, a slight lateral movement on the part of the patient introduces no appreciable error. The height of the patient's eyes above the tables makes no difference.

Details of construction. The woodwork can be made by any carpenter for a few shillings. The following measurements have been found satisfactory :—

The pedestal, 10 inches wide, 5 inches deep, from before backwards, $2\frac{1}{2}$ inches high.

The upright board, height $13\frac{1}{2}$ inches (11 inches above pedestal), width 5 inches, thickness $\frac{3}{4}$ inch.

The arm is pivoted at one end by a bolt which passes through the upright board. There is a chock on

each side, on one of which it rests. It is swung over to either side as required. This arm is of hard wood, 27 inches long, 2 inches wide, $\frac{3}{16}$ inch thick. It is painted black in front. A long strip of white paper or celluloid, about half an inch wide, has marked on it the tangents to degrees at the distance of 60 cm. This strip is pasted on the back of the arm, with the zero of the scale at the pivot hole.

The tangents to degrees at 60 cm. are—

2°— 2·1 cm.	18°—19·5 cm.	34°—40·5 cm.
4°— 4·2 ,,	20°—21·8 ,,	36°—43·6 ,,
6°— 6·3 ,,	22°—24·2 ,,	38°—46·9 ,,
8°— 8·4 ,,	24°—26·7 ,,	40°—50·3 ,,
10°—10·6 ,,	26°—29·3 ,,	42°—54 ,,
12°—12·8 ,,	28°—31·9 ,,	44°—57·9 ,,
14°—15 ,,	30°—34·7 ,,	46°—62·6 ,,
16°—17·2 ,,	32°—37·5 ,,	48°—66·7 ,,

In inserting the bell push only one wire is cut, the other being left intact. The instrument is made by Messrs. Bonnella and Son, 58, Mortimer Street, London, W.

Modifications of the Amblyoscope.

Dr. Ernest Maddox uses a quickly acting screw, instead of the short slot in the brass arc. This admits of delicate adjustment, which is an advantage in testing the horizontal breadth of fusion.

Dr. Freeland Fergus employs a method of illuminating the object slides which is certainly more compact than the apparatus shown in fig. 12, and is probably equally efficacious. "Immediately behind the parts of the Amblyoscope into which the pictures are placed, there is a stout metal collar from which a curved piece of metal is carried in front of the pictures. To each of the two pieces of metal an electric lamp of about three-candle power is attached. From each of the two lamps a lead is taken to a rheostat, so that the amount of illumination of each is thoroughly under control."

Dr. MAITLAND RAMSAY has substituted total-reflecting prisms for the mirrors ("Ophthalmoscope," vol. 3). These are, of course, very perfect reflectors, but very expensive. He has also devised an improved lighting apparatus. "The lighting is arranged by means of two small lamps, one fixed immediately

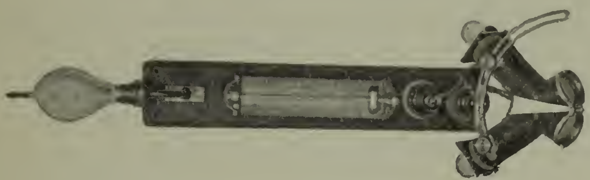

FIG. 41.

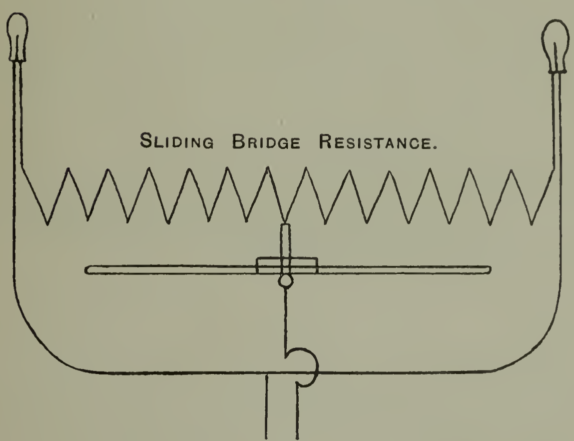

FIG. 42.

behind each picture in the Amblyoscope. The degree of illumination is varied and regulated by a sliding bridge-resistance, which can be adjusted most delicately and which, while it takes resistance from the circuit of one lamp and so increases its brightness, at the same time throws more resistance into that of the other and brings about corresponding diminution of its brilliancy.

The illustrations show the arrangement, which has been carried out by Mr. Trotter, 40, Gordon Street, Glasgow."

Dr. Nelson Miles Black has added a vertical screw adjustment, by means of which one tube can be moved above or below the plane of the other, in order more readily to adapt the instrument to cases of squint in which there is a vertical deviation. By means of this ingenious device, one may, in a case of obstinate suppression, call attention to the second image by temporarily throwing it above or below the plane of the other.

Advancement Forceps.

Messrs. Weiss and Son have made for me an advancement forceps on the Prince model, which seems perfect. There are no spikes, the blades are smooth and probe-pointed, so the instrument is as

Fig. 43.

easy to introduce as a tenotomy forceps. The catch gives a firm grip and has not the same tendency as the spring catch to get out of order. Only one forceps is required.

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